Inflammation of the Lungs:

Tuberculosis and Consumption.

BY

Dr. Ludwig Buhl.

INFLAMMATION OF THE LUNGS:

TUBERCULOSIS AND CONSUMPTION.

TWELVE LECTURES.

BY

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TRANSLATED BY PERMISSION FROM THE SECOND GERMAN EDITION,

BY

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TRANSLATOR'S PREFACE.

When Niemeyer first advanced his theories concerning the origin of Phthisis and Tuberculosis, he awakened renewed interest in a subject which had generally been regarded as settled. His views, so radically different from those received by the majority of the profession, attracted many advocates, and have been extensively adopted.

Many of the most active practical workers in pathology, however, have never given their unqualified assent to these views; and particularly has this been the case in Germany. Investigation and observation have thus been greatly stimulated; many new discoveries have been made and theories advanced.

Prominent among the latter are those advanced by Prof. Buhl, of Munich. These theories, the result of long and careful investigation and observation, both clinical and anatomical, coming from an active worker and experienced observer, are certainly worthy of the careful consideration of the profession. Particularly is this the case when we remember that Prof. Buhl is the author of the infection theory of tuberculosis, which was the groundwork and foundation of Niemeyer's structure. These views are not altogether recent with the Author, he having held them in part and taught them

for some time; they have, however, never been published before in a connected form.

Many of the German observers, who were not contented with the explanations offered by Niemeyer and his followers, have adopted the ideas advanced by Buhl, either fully or in part. Conspicuous among them stands the distinguished pathologist Rindfleisch, whose work is already familiar to American students of pathological anatomy. Others, although not adopting these views *en masse*, admit that they are of great importance; and that they are worthy of the most careful consideration and study.

We ask, then, from the profession in this country, the same consideration for Prof. Buhl which he has received at home; and hope that by renewed discussion and still deeper investigation the truth or falsity of these pages may be proven.

The book was published originally in the form of "Twelve Letters to a Friend." We have, with the Author's permission, changed this form to that of Lectures, as being more suitable for a scientific work.

NEW YORK, May, 1874.

PREFACE AND DEDICATION.

The four hundredth annual celebration of our University, the establishment of a pathological institute, and the twenty-fifth anniversary of the commencement of my teaching, occur almost simultaneously. I wish, therefore, in these pages to leave to the University, the celebration of whose birthday has for me a double interest — since my long-cherished desire is about to be fulfilled — an account of a few researches, incomplete though it be; and at the same time to dedicate to my medical friends and scholars a remembrance of my pathologico-anatomical demonstrations.

In the course of these demonstrations I treated by preference of pulmonary diseases; I have, therefore, concluded that I ought to choose the same subject in order to accomplish my present object.

These letters were written in the midst of a great war, in the excitement of which I accompanied the victorious armies and the trains bearing the dead. Unfortunately I was often hindered in their elaboration, and therefore they are, to my disappointment, much belated in their completion. Although originally intended only for a friend, I make them now accessible to a wider circle, and ask pardon if much in the

demonstration is not as successfully given as I could wish. From their peculiar form it will hardly be demanded that they should possess the completeness of a handbook or monograph.

May the offering be received with the same friendly sentiments which I have heretofore experienced; and may it be considered as the expression of my thankfulness for past kindnesses.

THE AUTHOR.

MUNICH, July, 1872.

PREFACE TO THE SECOND EDITION.

THESE Letters, which I published with some anxiety, have, contrary to my expectations, met with a favorable reception. This may be due to the important subject of which they treat; for what I have advanced needs much improvement and further investigation, and especially confirmation at other hands.

As every new structure can only after the lapse of time prove whether it can endure the severe trials of adverse circumstances, so years will go by before it will be acknowledged that the substance and structure of my views are firmly enough founded to take rank in science. Already many of my fellow-workers have surprised me with such commendations that I send this edition forth with less anxiety. I especially rejoice that Rindfleisch (Vortrag in der Sitzung vom 18 November, 1872, in der Niederrhein Gesellsch. für Natur- und Heilkunde in Bonn) not only supports my view of desquamative pneumonia, but also accepts most of my most important conclusions.

I could myself easily name the weak points of my work, did I dare hope that other investigators would help me to throw light upon these dark places. I would especially invite any such to direct their energies

to the histogenesis of tubercle, which is one of the most difficult of subjects. The subject of the nature of infectious material has recently come up in a new phase, and must exercise an influence upon the theory of tubercular infection.

As regards the desquamation of the alveolar epithelium, which can almost always be seen in a certain degree in the cadaver, investigators will gradually learn to distinguish between what I mean by genuine desquamative pneumonia, what belongs to other inflammations, and what is to be considered as indifferent desquamation.

The few additions which, since the first appearance of my work, I have thought best to introduce here and there, have been partly incorporated in the lectures themselves, and partly added as notes.

MUNICH, May 17, 1878.

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INFLAMMATION OF THE LUNGS:

TUBERCULOSIS AND CONSUMPTION.

LECTURE I.

GENTLEMEN:—

IN introducing the subject of Consumption and Tuberculosis, I may say that the investigator who shall enable us to comprehend the nature and causes of the above diseases to such an extent, that we can successfully cope with them, will confer a most signal blessing upon mankind. But thousands of years of applied industry are already gone, thousands of men of original talent have passed to their graves, and it seems as if no material advance had been made in the elucidation of the subject. Any important newly-found fact may overthrow the previously-erected theoretical structure and compel us to begin anew, and when we think we have surmounted difficulties and gained positions which will render our further progress easy, we find fresh and formidable obstacles in our path. If we think, for example, that we are on the point of finding out the cause of Tuberculosis, our results sink into insignificance when we turn to investigate the aetiology of Consumption, which appears to us like a true "*terra incognita.*" A long time must elapse before we shall be able to construct the terminal theory based upon the last possible acquisition. A short time ago light was first thrown upon Tuberculosis, and although Consumption is as old as the civilization of the human race and surely cannot have escaped the penetration of attentive observers, by reason of its fearful phenomena,

yet our knowledge of the *tubercle* and of the connection between Consumption and Tuberculosis (Laennec) is a work of the present, a result of pathological anatomy. Since the number of those who die of Consumption is very great, especially in the larger cities, we may imagine what a number of bodies have already been examined in order to obtain and verify the above truth (I mean the connection between Consumption and Tuberculosis). But after this fundamental principle was admitted in its grossest features, the results of examination were so uniform that we considered our investigation of the subject at an end, and finally, indeed, thought it useless to examine all those who died of Tuberculosis. Since I began to teach I have opposed this belief that we were long ago done with the pathological anatomy of Tuberculosis and Consumption, which was so common that we felt almost compelled to remonstrate if the old subject was again brought up, and I must observe that in no other region of pathology do so many obscure opinions prevail as in that of Consumption and Tuberculosis, and with reason, for the researches in this department are indeed of a difficult nature. Fortunately, however, at present Consumption and Tuberculosis have again taken a place among the scientific questions of the day.

I have endeavored to the best of my ability to arrive at the truth of this question and place it before you, yet I find that the presentation of what is already known offers great difficulties, and hence for the sake of a correct appreciation of the propositions which I shall advance I will first speak of the better-known inflammatory lung-diseases, a knowledge of which must precede in order to obtain for Consumption and Tuberculosis the necessary foundation and the proper point of view. I shall subsequently broach my own theme, the desquamative or parenchymatous inflammation upon which the study of the former may easily be engrafted. Some preliminary remarks, moreover, are necessary for the sake of calling your attention to certain anatomical relations.

If you search the inflammatory conditions of the respiratory organs, it will not escape you that there are forms which show the characteristic signs of their course superficially, in the inner, epithelial surface of the bronchi and lung-alveoli, while others give these signs more in the bronchial walls and the adjacent proper parenchyma of the lung. Since here the question is only one of degree you will find in the establishment of two groups, the *superficial* and *parenchymatous inflammations*, no absolute separation, and will not understand that a superficial inflammation has nothing to do with the lung-parenchyma, and that a parenchymatous inflammation does not implicate the surface of the bronchial system and the alveoli. On the contrary there is no superficial inflammation seated *only* in the epithelium without participation of the parenchyma, and no parenchymatous one without participation of the epithelium, only, as will appear, that which merely participates is the subordinate inflammation. In reference to the inner surface of the alveoli I must observe that I have always belonged to those histologists who accept as absolutely certain, the existence of an epithelium not only in the fœtal condition but also after birth and up to old age. My opinion coincides with that of F. E. Schultze (Strickers-ches Handbuch der Gewebelehre) whose description indeed, obtained from careful observation, solves the most doubtful questions concerning the minute structure of the lung tissue. In the explanation of the alveolar epithelium, however, I must say, that I consider this to have far less the characteristics of a continued bronchial epithelium than of a *lymphatic endothelium spread out upon the inner surface of the alveolar wall.* This view is sustained by the fact that the shape and size of the cells are entirely different from those of the bronchial epithelium, while on the other hand, the resemblance in shape to that of endothelium is scarcely to be mistaken, as Ranvier has already insisted. The lack of continuity too, observed by many histologists, finds an analogue only in the endothelium of a serous surface. The bronchial epithelium consequently passes over into the

alveolar endothelium, as the tubal epithelium into the endothelium of the peritonæum. The experiments of Sikorsky (Centralblatt. 1870. No. 52) furnish a convincing proof of this, since from them it appears that there exists in the alveolar wall, a plexus of lymph vessels consisting of canals and stellate connecting-knots, and that these knots communicate by means of fine openings with the cavity of the lung-alveolus. Some may consider the alveolar cavities as broad lymph-spaces containing air, and having a lining endothelium, the latter being wanting in the lymph-plexus of the alveolar wall. As regards the respiratory interchange of gases this relation cannot be without significance, since no absolute obstacle in the wall of the alveolus is placed in the way of the inspired air, which may be admitted into the blood, not only through the capillary vessel by diffusion of gases, but by direct entrance into the open alveolar lymph-plexus.* The lymph-gases, whose origin, as yet, whether from the blood or the tissues, is unknown, and which according to Hammersten (Bev. d. sächs Ges. d. Wiss 1871 Oct. 617–634. Ueber die Gase der Hund-lymphe) show the significant proportion of 20–40 per cent of CO^2, may be furnished, in great part, through direct taking-up of inspired atmospheric air, and quicker change into CO^2. Finally, there is a remarkable analogy between the alveolar epithelium and the lymph-vessel endothelium in pathological processes, especially those of a productive nature, an analogy which I remarked long ago, and to which I shall refer hereafter. These are, in brief, the reasons which lead me to a conception of the alveoli and their epithelium different from the common view.† When I

* If the air taken up by the lymph is as invisible as that in the blood (because incorporated at once with it) the interlobular emphysema of very dry lungs (*eg.* in Cholera) may be nothing more than atmospheric air pressed into the lymph vessels in great quantity and in visible vesicles.

† Friedländer opposes my view in his splendid researches upon pneumonia which have just appeared. His first grounds of opposition are that a lymphatic network existing beneath the alveolar wall and communicating with the lumen of the alveolus in no way

now speak of superficial inflammations I mean those forms whose products are shown especially in the epithelium, or which run off over its surface and consequently reach the cavities of the alveoli and bronchi, while the products of the parenchymatous inflammations appear as interstitial and peribronchial.

The vascular apparatus also, of the respiratory organs is peculiar. As in the liver, a vascular trunk enters from two sides, one arterial, and one (by the nature of the blood) venous, so as to meet in one common capillary plexus, so in the lungs we have a like condition. I do not say this to institute a comparison, for the differences are significant and manifest at first sight, but I mention it in order to bring forward prominently the fact that not only the *pulmonary* arteries, but also the *bronchial* arteries, which commonly are left out of account, take part in the capillary circulation of the lungs, and therefore also in the disturbances of the nutrition of their tissues. It is proven, however, that the bronchial arteries run more in the interstitial and interlobular tissue and, finally branch in the pleura, while the

necessitates the lymphatic nature of the alveolar epithelium, and moreover, that neither for the alveolar epithelium nor for the lymphatic endothelium is an analogous relation insured in pathological formations, and finally that he cannot admit the lack of continuity of the alveolar epithelium. I pass by these objections at once, since they are of a negative nature. A more important objection is, that the facts in the history of development testify strongly to a simple continuation of the bronchial epithelium, and that there is not the least doubt that in the fœtus the inner layer of the alveolar wall consists of true epithelium. This is the turning-point of the whole question. To this I would reply, that according to my observations up to the present time, the inner layer of the alveolar walls does not consist of true epithelial cells, and that both embryonal testimony and anatomical relations show that the epithelial formation ceases at the bronchioles, *i. e.* at the beginning of the infundibula. (See also die Anm. p. 68,) Whether Debove (Compt. rend. LXXV, No. 26, 1872) was right in recognizing beneath the bronchial epithelium an endothelium which alone is continued into the alveoli must be determined by further investigation.

pulmonary arteries, which are of much greater diameter, form the principal mass of the alveolar capillaries. From this relation it follows that the peribronchial, and the parenchymatous inflammations occur especially within the domain of the bronchial arteries, while, on the other hand, the superficial inflammations of the lungs, (not of the bronchi) take place within the region of the pulmonary arteries. Furthermore, on the one side, appears the sympathy of the pleura and bronchi in primary inflammation of the parenchyma of the lung, as well as the possibility of the spread of a disease of the bronchial branches and of the pleura to the lung-parenchyma, and on the other the actual regularity in the mode of appearance of the one or the other of the above diseases. For instance, primary diseases of the lung-parenchyma have, like the capillary circulation, a diffuse, continuous spread, *i. e.* lobar ; on the contrary, primary disease of the bronchi will nearly always leave certain branches free, and if the mischief spreads to the lung-parenchyma it will always appear in a lobular form, at least at the outset, *i. e.* the diseased lobules will have near and around them, those which are relatively healthy or only slightly diseased.

My definition of *lobar* and *lobular* then, does not, as is usually the case, depend upon the *extent* of a disease in the lung, but rather upon the manner of origin and spread, and the position in the tissues.

We see in thrombi and emboli how the double vascular apparatus governs the double phenomena. The ordinary thrombi occur in the capillaries fed by the pulmonary artery, which are generally greatly dilated, and produce therefore lobar masses (infarctions of Laennec). The so-called "pyæmic wedges," dependent upon emboli in the finer pulmonary arteries, however small they may be, are none the less definitely lobar in situation and spread. If we have large infarctions (of Laennec) or foci as the result of emboli there can be no doubt concerning them. In contrast to this, there occur, although seldom, in connection with atheroma of the openings of the bronchial arteries into

the thoracic aorta, diseases of the lungs dependent upon anæmic necrosis, (previously considered as Tuberculosis) which bear the most indisputable lobular character as regards situation, origin and progress. The *number* of diseased foci is as unreliable as the size to employ for a distinction between lobar and lobular, for as there are single lobular spots, so are there multiple lobar masses although commonly the reverse is the case.

The diseases which proceed from the lymph-vessels stand, to a certain degree, undetermined between these two forms, the lobar and lobular. Since the lymphatics, from their finest origin in the alveolar walls, follow the course of the bronchial arteries and run as interlobular, interstitial and peri-bronchial, to collect at last, on the one hand in the subserous pleural envelope, and on the other, in the root of the lung, their affections may be, in one case, more continuous and diffuse, in another, in small spots or limited to points, while a third time, both relations may coexist, and we may doubt whether it should be called lobar or lobular. Indeed, there are at times in this region, allied diseases which are spread diffusely over both lungs or one entire lung, or are confined to lobes or lobules.

You will find a final difference, not only at the sick bed, but also anatomically, when you study inflammatory conditions with due regard to the rapidity of their invasion and the time they have existed. Acute and chronic diseases have different characteristics. The superficial and parenchymatous, the lobar and lobular inflammations appear otherwise, if the changes in the lung tissue which occur in their course, have originated suddenly or rapidly and at the time of the examination have only existed for a short period, than if they had developed slowly and through a considerable interval until the day of death or of examination. The latter are distinguished from the former especially by means of extensive and significant degenerations if, or in so far as, they were superficial, and by means of increase of connective

tissue, formation of cicatrices, etc., if, or in so far as, they were parenchymatous, interstitial or peribronchial.

I hold the three points mentioned as sufficiently important to serve as a basis in the observation of the inflammatory conditions of the lung. For all cases, however, in which secretions remain lying in the air-passages, or tissues are destroyed, it must not be forgotten that the contact with the air, which is peculiar to the respiratory organs, acts either directly or indirectly in a destructive manner upon the aforesaid substances, and thereby influences the whole body Commonly "Schizomyceten" or Spores are produced which may enter the blood or lymph-vessels from the putrescent spot.

LECTURE II.

Catarrhal Pneumonia.

We begin the series of inflammatory diseases with the "acute catarrhal pneumonia" of the books, which, however well-known and much used its name may be at the sick bed, is itself, in spite of the excellent observations of Bartels and others, as yet too little known, anatomically, so that many errors exist concerning it, which I will endeavor to correct. Rokitansky describes it, as follows: (III Bd. p. 15) "Not seldom the fine bronchi and the bronchioles are the seat of an especially active inflammation which soon spreads to the substance of the lung. This appears in lobular spots, commonly numerous, dark red, swollen, hepatized or filled with pus." I add to this that it is an acute disease, that it especially attacks the lower lobes of the lungs, particularly posteriorly and towards the edges, and only spreads upwards and forwards after long duration. The definition given, short as it is, contains all that distinguishes the so-called Catarrhal Pneumonia from other inflammations of the lungs, and, indeed, it may be so strictly applied, that a process which does not show the characteristics mentioned, or shows others, does not deserve the name of Catarrhal Pneumonia, but loses the signification attached to that name at the present time.

As we are now speaking of Catarrhal Pneumonia, we must understand that catarrh may have its seat not only in the fine bronchi and bronchioles, but also in the lung-tissue. Acute catarrh is a superficial inflammation with œdematous swelling and reddening of a mucous-membrane in whose epithelium it is especially seated, and the inflammation induces

not only an increase of secretion, but a change in it, so that to the mucus, there is added at the outset an excessive proportion of serum, often even of plasma with blood corpuscles, which is however, only temporary. Later, on the other hand, an excess of mucus, or pus-corpuscles is added, while there is a decrease of the exuded serum, this coinciding with a diminution of the œdema of the mucous-membrane. Now, since there is no mucous-membrane in the lung, the existence of Catarrhal Pneumonia has already been called in question. Oppolzer. (vid. Vorlesungen über, spec. Pathol w. Therapie. herausgegeben von Ritter von Stofella. I. p. 607) and previously Niemeyer, likewise felt, that, for the reason given, the term had not been aptly chosen. And, indeed, although, after all, the alveolar wall is nothing else than the prolonged inner fibrous layer of the bronchi (mucous-membrane), yet it is so completely reduced that it no longer in any respect resembles a mucous-membrane, and the epithelium lining it has undergone such important changes of form, that it can no longer be identified with the bronchial mucous-membrane. You will remember that, in my first letter, I considered the alveolar epithelium to be lymphatic endothelium. So then, the process, which appears as Catarrh upon the mucous-membrane of the medium and finer bronchi, fills them with mucus and pus, and in which the alveolar parenchyma of the lung has some share, can be considered as carried over only in a very modified form; it ceases not only to be a catarrh in the ordinary sense, but, as we shall see, to be a primary inflammation. If we, then, examine closely an affected lung, we recognize a dissimilarity of texture. In one part are spots which by reason of their excess of blood, are marked upon the pleural covering by ecchymoses, and are of a dark red color. These spots, being swollen, project, and from their cut surface flows freely not only an excess of blood but also serum mixed with air-bubbles (acute œdema of lung). In these places, and especially on the edges and towards the root of the lung, we find alveoli collapsed, retracted, almost or quite empty of air and of a

bluish red color (atelectasis). Again, in other lobules, in strong contrast, we find alveoli markedly distended with air, pale, and having blood only on their circumference, shut in, as it were, by a crown of injected vessels (local emphysema). Finally, we find, especially in chronic cases, lobules—and these are the ones which are particularly claimed as inflamed—having a smooth, non-granular section, a firm consistence, hence projecting above the cut surface, and a mottled, pale-yellow color. These in fact, owe their appearance to the filling of the alveoli, not with fibrinous contents but with thick *mucous* secretion rich in *pus-corpuscles.* The immediate surrounding of these centres is marked by hyperæmia. The microscope shows in them, aside from the masses of mucus and pus-corpuscles, epithelium in unusual quantity and in a state of fatty degeneration. The same is found also diffused in the œdematous spots as a sign of the peripheral prolongations of the inflamed bronchial tract. In contrast to this the emphysematous parts and the atelectasic tissue when artificially distended show scarcely any abnormality in their histological nature. The changes, which I have here described and which sometimes alternate, not only with each other, but also with perfectly normal lung-tissue and hence appear in a lobular form, sometimes are scattered over whole lobes, or the greater part of them, without leaving sound parenchyma between the altered parts, and, in this case, the lobular nature is shown in the wavelike section. They can be clearly distinguished only in cases of short duration; in the more chronic cases, œdema and emphysema are generally less marked, but rather give place to atelectasis and purulent engorgement so that finally an entire lobe appears by infiltration blue or brownish-red. The last-mentioned characteristic is especially shown in catarrhal pneumonias in which the element of hypostasis enters *e. g.* in typhus (our typhoid).* Among

* The conditions described by Friedländer (l. c.) after section of the pneumogastric give the appearances characteristic of the so-called hypostatic pneumonia as it may be seen in all diseases attended with

the changes, indeed, only the filling of the lobules with mucus and pus can lay claim to belong to the catarrhal process. The œdema of the lung is simply a collateral phenomenon of capillary bronchitis, and atelectasis and emphysema have nothing to do with inflammation. The bluish-red color of the atelectasic lobules signifies only the hyperæmia of the capillaries as this must occur with the cessation of the intra-alveolar pressure of the air, just as, on the other hand, the paleness of the emphysematous patches shows the increased pressure of the air, in the corresponding parts.

Nor can the the spots filled with pus be unhesitatingly considered as an expression of a lobular pneumonia, since for this end, the lobuli must themselves have produced their contents. The presence of pus-corpuscles offers now no difficulties to the explanation, for there is no doubt that, under changed relations, such as inflammation establishes, pus-corpuscles, *i. e.* white blood-corpuscles, may go directly through out of the alveolar capillaries. It is different, how-

considerable feebleness of circulation. The causes of hypostasis may alone cause the exudation of colored and colorless blood corpuscles (pus-corpuscles) from the vessels, and so bring about an infiltration of the lung parenchyma, or they may simply be superadded to already existing inflammations of the lung of any kind—most often catarrhal and consecutive pneumonia (mixed hyperæmia of Henle)—We are not justified in classing the hypostatic conditions in the lung with desquamative pneumonia, because there is desquamation of epithelium, and Friedländer himself has pointed out the distinction (p. 30. seiner. cit abth). Nevertheless he has plainly though erroneously, identified catarrhal with hypostatic pneumonia. Not to mention the difference of origin and possibility of the two diseases co-existing, the common element (pus-corpuscles in the lung-parenchyma) is far outweighed by the differences, viz. that in catarrhal pneumonia, it is not only the posterior and lower parts which are attacked as in hypostatic, but also the anterior and upper, that in contrast to the fibrine which appears here and there in hypostatic pneumonia, mucous bronchial secretion is an essential part of the catarrhal; that catarrhal pneumonia is a lobular affection, while hypostatic pneumonia is diffuse, and catarrhal pneumonia never occurs associated with pleurisy, while this is seldom absent in hypostatic pneumonia.

ever, with the mucus in which they are imbedded, for data are wanting to show that the alveolar epithelium, which has the physiological function of transmitting gases, of absorbing atmospheric oxygen, and separating carbonic acid and watery vapor, can also produce mucus, if we may not at once deny it this power. It remains, then, doubtful whether the muco-purulent contents of the lobules is formed in the lobules themselves; indeed, it is, on the contrary, certain (and indeed proven through the co-existence of groups of lobules, which are simply œdematous, with those which are atelectasic and emphysematous,) that the major part of the catarrhal product lying in the bronchi, if not all, springs from the bronchi and by movements of aspiration is conveyed thither, whence it is not removed by reason of a simultaneous closure of the corresponding bronchiole. Yet, although the collection of pus in the lobules of the alveolar parenchyma is apparently not a direct inflammatory product proceeding from it, we are not to suppose that subsequently if the disease continues the lung tissue may not be aroused to inflammatory irritation. From the description given, it follows that *that which is called catarrhal pneumonia is no pneumonia, but only a capillary bronchitis*, a *bronchiolitis in which the lung shares in the way of collateral œdema, atelectasis, local emphysema, and engorgement in consequence of the secretion being transferred from the bronchi to the individual alveolar lobuli.* It can be easily understood, however, that the disease, in its more significant degrees of severity and extension, may cause death with the symptoms of dyspnœa and cyanosis. If the bodily strength is sufficient, the extent of disease limited, and the implication of the lungs slight, it will almost always go on to a rapid and perfect recovery. This favorable issue has its basis in the fact that the parenchyma of the lung, aside from the relation mentioned, remains intact. Healing occurs through expectoration of the plugs from the bronchi, fatty degeneration and subsequent absorption of the purulent collections which have found their way into the alveoli, through absorption

of the serous infiltration in the parenchyma of the lung and in the bronchial mucous membrane, dependent upon renewed and more active capillary circulation, through retrogression of the local emphysema as a result of free entrance and exit of air, and through regenerative reparation of the small amount of epithelium which has been destroyed. If the disease does not prove fatal, but the bodily strength is not able to effect a quick and complete cure,—and this happens especially in weak children, in the aged, or in the course of severe diseases (typhoid, measles, etc.), where the strength of the inspiratory muscles is not sufficient to overcome the resistance—then isolated spots may, in consequence, be markedly noticeable, as not keeping equal pace with the resolution in the remaining lung-tissue, but lagging behind or even leaving a permanent residuum. I speak here not only of the local emphysemas which point to bronchi still occluded, but also of the atelectases, which only take up air again at intervals and sparingly, or remain permanent, and by virtue of degeneration of the enclosed epithelium, are changed into pigment or cheesy-centres (as in congenital atelectasis), and I also refer to the mucous and pus plugs in the finer bronchi and alveoli. The latter having become fixed, degenerate, and, as cheesy masses, obstruct the bronchiole and the corresponding lobule. There is, however, a striking proof which shows that cheesy degeneration in acute catarrhal pneumonia is not "tolerably common" (Niemeyer), but, on the contrary, must belong to the rarest of all appearances, and that is, that the cheesy centres found in the lungs occur very generally in the upper lobes and their apices, while catarrhal pneumonia would have to deposit its cheesy spots in the lower lobes; and moreover, they form only the contents of the bronchioles and are never penetrated by lung tissue, this itself taking part in the cheesy degeneration. The bronchiole surrounding the cheesy mass is enlarged like a vase and thickened, and the mass lies, as it were, in a fibrous capsule. The process of encapsulation is, then, never the product of the catarrhal inflammation of the mucous mem-

brane, but the expression of the activity with which the whole bronchial wall, its connective tissue layer, and the adjacent alveoli respond to the irritation of the foreign contents; the product of a chronic peribronchitis.*

This irritation may, of course, at one time, be degenerating pus and mucus, but, at another, as we shall see hereafter, some other foreign contents. Indeed, it is often difficult to decide whether the process did not begin in just the reverse way as interstitial or peribronchial, and the catarrh of the mucous membrane with its products is not the *result.* Moreover these encapsulations occur almost exclusively in the apices of the lungs, and we must be careful how we refer them to acute catarrhal pneumonia, yet they could, at least if we admit a chronic bronchitis as an exciting cause, be the result of a deficient expulsion of bronchial secretion dependent upon increasing bodily weakness (*eg.* in the bed-ridden, in sufferers from cancer, etc.), which secretion in the continuous horizontal position is most difficult to dislodge in the upper bronchi. But to trace back the encapsulation to chronic bronchitis as a process, not as a cause, instead of to peribronchitis, is to mistake the situation and progress of a catarrh. In addition, the most protracted catarrh is limited to the mucous membrane, thickens only the innermost layer of the bronchial wall, and leaves the other layers, especially the connective tissue bronchial sheath, unaffected. Need I specially adduce that the cheesy, encapsulated mass, which is of a ring-bullet, or spherical form, which continually tends to become dryer, and later on becomes chalky and stony, and which is of a size from that of a pin's head to that of a pea was formerly considered as tubercle in the apices of the lung? (encysted tubercle). I believe that no one nowadays falls into this error, and every student knows how cheesy thickened pus and tubercle are to be distinguished from each other. It would be just as erroneous to place these plugs of cheesy matter on a par with the characteristics

* See Letter VIII.

of cheesy pneumonia, from which a whole series of false conclusions might arise. But more of this hereafter.

The foregoing observations, especially the derivation of the lobular purulent centres from a transference of the muco-purulent secretion from the finer bronchi to the alveoli, lead me to speak briefly of other more peculiar foreign bodies forced into the respiratory tract through respiration, and of their inflammatory products. In many respects, the changes induced by foreign bodies so long as they are superficial, correspond with those which we are wont to call catarrhal pneumonia. It is self evident, that, by this, are not meant such foreign bodies as by virtue of their considerable volume remain impacted in the larger bronchi whose walls and even large pieces of the corresponding lung tissue perish by gangrene. I mean particularly *smaller* bodies, which by force of aspiration are drawn into the smallest bronchi and the alveoli, just as happens with the mucus and pus in capillary bronchitis, and which, in the place of their final lodgment, give rise by continued irritation, to superficial epithelial disturbances, indicated by desquamation and degeneration.

To name then, without delay, such intruders, which in like manner, have been produced in the mucous membrane of the upper respiratory tract and of the pharynx, I mention blood extravasated from the vessels of the mucous membrane, and croup of the larynx and of the trachea in which pieces of the membrane are drawn in till they reach the lung parenchyma. Diptheria offers equal interest, a process which appears, as is known, in the pharynx and spreads to the laryngeal and tracheal mucous membrane, and as I have shown (Zeitsch für Biologie III., page 357), also manifests itself in lobular centres in the lung-parenchyma. Recently I found in the latter, besides the diphtheritic infiltration, a little heap of long ciliated cells cast off from the upper part of the respiratory tract. But also, without hemorrhage, without recognizable croupous diphtheritic affection in the upper air-passages or in the pharynx, there occur in rare

cases, exactly the same foci in the lung tissue, which can be distinguished from the lobular purulent centres of the so-called catarrhal pneumonia by the fact, that not only the contents of the alveolus shows the purulent quality, but also that the alveolar, and adjacent bronchial-walls are friable and of the color of yellow pus, which change is produced by a growth of the vessel; and tissue-nuclei, just as I have described it in lung-diptheria. · In severe cases of epidemic influenza, in measles, etc., I have noticed this remarkable relation. The spots have something quite peculiar and characteristic, which is warranted by the fact that they contain a nest of "Schizomycecten" or "Pilzen." Their vicinity is hyperæmic and full of extravasations which are the cause of the bloody sputa discovered at the sick bed. The so-called putrid bronchitis which is commonly seated in the larger bronchi may, likewise, through transference of its products into the alveoli, occasion putrescent destruction and give rise to lobular foci. Here also is to be classed *pneumonomycosis*, and not only the well known *pneumonomycosis sarcinica*, or *aspergillina*, but also the form which tolerably often appears in the later stage of typhoid, giving rise to small gangrenous-smelling spots of dark and bloody appearance in which, not only within the terminal bronchi, but also in the contiguous lung-parenchyma (hence lobular), *Zoogloeawassen*, terminally growing threads and true Pilzen in great quantities, indicate the peculiar disease. We should also note here contents of cavities transferred from the lung itself, and we may add the more rare instances of particles of cartilage being wedged into the alveolar parenchyma in cases of necrosis of the larynx, and also the large easily recognizable cells of epithelial cancer, (of the larynx, the tongue, the œsophagus, with perforation into the trachea).

In ileus, death usually occurs before the reacting appearances in the lungs show themselves, appearances due to particles of excrement which are to be found in the mouth and from thence are inspired. Yet these organs often show clearly enough the effect of the foreign masses, and with the

microscope, we can recognize in the lobular centres bile-stained pieces of muscle, vegetable fragments, starch, etc.

Very recently the diseases caused by inhalation of dust ("pneumonokoniosen" of Zenker) have excited great interest, especially those caused under certain circumstances, by particles of chalk, flint, clay, coal, metal, or manufactory-dust of different kinds, suspended in the atmosphere in considerable quantity, which substances make their way into the lung-parenchyma where they may be microscopically and chemically detected. Zenker, in particular, has acquired a lasting reputation by his critical establishment of these diseases formerly suspected almost solely in reference to coal, but which have now acquired a very extensive application. Finely divided bodies remain often clinging only to the epithelium of the bronchi and alveoli, and may cause their destruction and desquamation, or even catarrhal bronchitis and lobular pneumonia. Small bodies, such as iron, coal, flint, chalk, etc. are taken up, not only by the superficial cells, but also by the lung-tissue itself, less often by the bronchial tissue, and there is no difficulty in explaining this process, especially in the lung-tissue, in my conception of the alveoli as lymph-spaces. They are then further transported by means of the tissue fluids or migratory cells to be deposited either on the way or in distant parts, *e. g.* in the bronchial glands. It is certain, that this deposition may take place without especial, indeed without *any*, irritation of tissue, such a case we have in mind at present. It is, nevertheless, just as certain that, by means of it, important and dangerous conditions may be induced. In this latter unfortunate direction, the previously described organic substances found in putrescence and abounding in fungous and fungoid organisms, act in an especially acute manner, since they cause rapidly a deeply-seated lung disease and even blood-infection. Inorganic dust-particles attack more chronically, and particularly affect the interstitial and peribronchial tissue, these cases therefore no longer belong under the head of superficial inflammation of mucous membrane. In a typical case

of acute process the lung has a peculiar appearance. It is, by preference, the lower lobe and generally the right lung which is affected. It is bulky, succulent, friable, filled with dark blood, full of extravasations and of a green or brownish-black color. We recognize foci from the size of a pin's head to that of a walnut, having retained air, pus, and a gangrenous odor. If we consider as a result of catarrhal pneumonia, destruction by ulceration and gangrene (Rokitansky a.a.O.), I am quite convinced that these processes are always a consequence of foreign bodies which have gained admission to the finest bronchi and alveoli (pneumonia from foreign bodies) and have nothing to do with catarrhal pneumonia, or only in so far as the bronchial mucous membrane is irritated at the same time. You see then that I have materially narrowed the field of catarrhal pneumonia. I hope, however, that I have not only stated what is true, but that I have discovered a foundation for the study of the other inflammatory conditions of the lung.

Catarrhal pneumonia, according to my conception, is an acute disease by which local emphysema, atalectasis or mucous plugs in the bronchi are occasioned only in exceptional cases, and which has scarcely any other sequelæ. But I must not conclude without referring to one chronic result of catarrhal pneumonia, although it is quite rare, viz., *Atrophic alveolar and bronchi-ectasis.* It is seen in both the lower and upper lobes and gives the lung the appearance of a coarse-meshed sponge. The bronchi—mostly those from the fourth division downward,—are enlarged cylindrically or varicosely, the enlargements being at short intervals, or they may even be sacculated. Their mucous membrane remains intact and is covered with ciliated cells even in the sac-like swellings, and they contain secretion in greater or less quantity. The affection of the small bronchi often appears relatively or even absolutely greater than that of the larger, the dilatation increasing often toward the parenchyma. Upon section, especially if the lung has been previously distended and dried, we see round or oval spaces filled

with air, varying in size from that of a barley-corn to that of a walnut, and even attaining to the size of a hen's egg; in one place, not differing much from each other in diameter, in another varying greatly. The proper alveolar parenchyma has, for the most part, disappeared, and with the microscope we may distinctly see the gradual diminutions of the alveolar junctions and the formation of larger spaces out of the infundibula. The latter condition, therefore, lung-emphysema accompanies, more or less, the bronchiectasis.

If it were desirable we might establish variations of Emphysema and Bronchiectasis upon the differences which we find, but we should not by this accomplish anything scientific or of practical value. A closer search reveals fatty degeneration or entire absence of the alveolar epithelial cells, besides obliterated capillaries and in all probability obliterated lymph vessels, and pigmentations in their places together with fatty degeneration of the muscles of the bronchioles and of the alveolar parenchyma. There is no doubt that the whole process is brought about by gradual dwindling and absorption in consequence of constriction and obsolescence of the capillaries, and the name "atrophic" should never be forgotten, to distinguish it from other varieties of bronchiectasis, such as we find in peribronchitis.*

Old age furnishes the necessary conditions for the formation of this alveolar; and bronchiectasis after catarrhal pneumonia. The condition may however, develop in childhood, which is indeed most liable to attacks of catarrhal pneumonia, and especially in whooping-cough, of which this disease is the anatomical foundation.

Paralysis and fatty degeneration of the bronchial muscles by reason of inflammatory processes in the bronchial mucous membrane, adhesiveness of secretions, pressure of these secretions and of the air retained in the alveoli upon the capillaries, causing obliteration and degeneration of the latter, give rise to the disappearance of the parenchyma framework, and ectasis in consequence of the equally persistent stretching

* See Lectures VI. and VIII.

of the pleura. The remaining capillary plexus of the lung becomes wide meshed and not only loses in extent, but gives up more or less the character of a respiratory surface. Fortunately this condition, at least in its higher grades, is rare, and it is noteworthy how when acquired in childhood, it may be carried for a long time, often even to old age. It stipulates no phthisis.

The bronchiectasis dependent on compression of the lung in consequence of pleuritic exudation is, of course, not to be confounded with the atrophic form, any more than that connected with peribronchitis. The atrophic bronchiectasis is generally on both sides, and also in the upper lobes, that following compression, mostly on one side, and only well pronounced in the lower lobe ; the atrophic form shows spongy lung tissue dependent upon ectasis of the alveoli and bronchioles, while after pleuritic exudation the alveolar parenchyma and the bronchioles are compressed and useless, and the larger bronchi are enlarged. In atrophic ectasis, as a rule, pleuritic adhesions are wanting, while they give to the other its characteristic. Both have, however, in contrast to peribronchitis, absence of infiltration in the bronchial wall, provided that the affection is uncomplicated.

LECTURE III.

Croupous Pneumonia.

CROUPOUS pneumonia, the subject of the present lecture, is so well known, that I may omit many details, and need say only what subserves the purpose at which I aim in my lectures. Coupling it with catarrhal pneumonia (capillary bronchitis) I might at first bring forward the similarities and differences existing between these two diseases.

Croup of the air passages is an intensely acute form of inflammation. Writers have always avoided speaking of a chronic croup, yet if it does occur, (*e. g.* in the cases where branching tree-like bronchial casts have been coughed out,) they were wrong, for we must confess that such a course would only lead to confusion. I prefer the term "fibrinous bronchitis" used for this by Lebert,* in order to preserve the idea of croup, be the etymology of the word never so unfavorable.

Croup is, then, an entirely superficial inflammation, and its exudation is to be considered, like that of catarrh, as a product of secretion, with this distinction, that the normal secretion is changed, not only by transudation of blood-serum (catarrh), but of fibrine. This fibrine wells up above the epithelium† apparently as happens with normal secretion, and in spite of the rapidity of the process, none the

* Deutsches archiv für klin Med. VI.

† It is inconceivable to me why we continually read that the epithelium is lifted off by the exudation beneath. The examination of any recent croup can satisfy us on this point. It is only in the later stages when the exudation is over that the epithelium is lost through degeneration.

less with active participation of the latter, and coagulates soon, together with the corpuscles, which, as a medley of red blood disks and especially of cytoid (lymph—or colorless blood) corpuscles are at the outset imbedded in small quantity in the tolerably adhesive fibrine, but which gradually appear in greater quantity, and at the expense of the adherence of the fibrinous clot. The epithelium beneath the clot suffers no further change than swelling, cloudiness and commencing fatty or mucous degeneration, but we may often find pus corpuscles lodged in many of the cells.

You will remember that I called your attention to this occurrence of pus-corpuscles in epithelial cells in croupous pneumonia* and expressed the opinion that the former could be formed in the latter, describing this probable process as a free formation, occurring independently of the cell nuclei, and taking its origin from within, from the protoplasm, after the manner of the segmentation of the vitellus. This view, although adopted by many, was abandoned in consequence of the deductions of Cohnheim, and the general opinion at present is in favor of the view that all pus-corpuscles have wandered through the vessel wall out of the current of blood. I do not, of course reject this view in face of the proofs adduced, yet I cannot at once admit that migration from the blood is the only source of the pus-corpuscles, and I await a sound explanation of the fact that pus-corpuscles occur inside of the cell cavity not merely invaginated, but actually in the protoplasm of perfect epithelial cells with shut walls, which enlarge correspondingly, for although this may not coincide with the theory of Cohnheim, it is nevertheless correct and is not to be ignored and set aside in silence.† Yet, I will not delay you with the details of this

* Virch. Arch 1859. YVI. p. 168.

† The most recent experiments of A Böttcher show decisively, that it is possible to excite a central keratitis which does not develop from the periphery of the cornea, but arises in the irritated centre itself, so that the pus-corpuscles which are to be found in the centre, cannot have wandered in from the edge of the cornea, but must have originated in the spot itself.

question, it is my purpose only to sketch the nature of the croupous exudation, and I therefore again call your attention to this, since with my views concerning the nature of the alveolar epithelium, I must hold somewhat unusual opinions about croup of the lung, and this is, essentially, that the exudation is more of lymph than of extravasated blood.

If we now compare the croupous pneumonia with the catarrhal, we find this point in common, that both are acute and superficial inflammations, but at the same time we recognize an important difference. I have described the changes in the lung tissue in catarrhal pneumonia, as secondary, passive and proceeding from the bronchi, and as a result of this they always spread in a lobular fashion, but on the contrary croupous pneumonia has its seat primarily and actively in the lung tissue and hence is always diffuse and lobar, and spreads to the bronchi only secondarily. This relation, which harmonizes with the nature of croup-exudation, gives rise to further considerations.

The initial stage of croupous pneumonia is called the stage of *inflammatory engorgement* (*engouement*). If croup were an exaggerated Catarrh as I have elsewhere intimated, this preliminary stage would be a true catarrhal pneumonia, distinguishable from the previously-described lobular form, by its lobar, diffuse nature. But the name "catarrh" belongs to the anatomical seat, the mucous membrane, rather than to the special manner in which the disease progresses. It will, therefore, be better to consider the situation as reversed, and to say that, as we see that in catarrh, serous exudation is mingled with the normal secretion of the mucous membrane, so, in croup of the lung-alveoli, the same addition to gas and watery vapor exists, but sometimes the serum is replaced by perfect lymph.

Viewed in this light, the inflammatory engorgement of the lung parenchyma (together with the enlarged capillaries distended with blood, and small extravasations of red and white blood-corpuscles in the alveoli) is indicated by transudation of blood-serum in a manner quite analogous to

catarrh of the bronchial mucus membrane, and if the exudation gradually comes to contain more and more fibrine, the process may be looked upon as croup.

The inflammatory engorgement is announced, as is well known, very suddenly (with a chill) and proceeds after a short duration (generally in less than one day), directly to hepatization, *i. e.*, to the condition in which the croup exudation fills the alveoli and bronchi as a clot. It fills up these spaces in succession, step by step and without intermission, and it is so abundant that the air is completely excluded.

The characteristic sign of croupous pneumonia is thus reached. The parenchyma swollen with serum compresses the coagulated contents of the alveolus as firmly as it receives pressure from the latter, and hence upon the cut surface, the croupous plugs, these casts of the infundibula and alveoli, are driven forward as detachable granules. And since the red blood extravasations are perhaps still more numerous than in the stage of engorgement, the capillaries must suffer a diminution of their calibre, although not losing their circulation. The whole diseased lung has experienced an increase of volume and weight, the investing pleura is stretched and likewise swollen, and the friability of the lung-parenchyma is communicated also to the pleura in consequence of the loss of elasticity. The pleura becomes not only clouded, through loosening of its epithelium, but also covered with transuded fibrine (lymph-coagula, croup-membrane), analogous to the exudation upon the inner surfaces. This fibrinous pleurisy, which is a constant accompaniment of croupous pneumonia (pleuro pneumonia) is wholly wanting in catarrhal pneumonia even in lobular spots. I pass over the fact that in some cases considerable quantities of serum are poured out into the pleura. The hepatization described, which, by reason of the visible exudation (austritt) of red blood-corpuscles, has received the appellation "red," is the essence of croupous pneumonia. Any one who has had considerable experience in the typical course of this form

of inflammation of the lung, knows that generally on the seventh day, the symptoms (rise of temperature, frequency of pulse and respiration, dyspnœa and cyanosis) diminish almost as suddenly as they appeared. The whole space of time of seven days, of which about one or one-and-a-half belong to the period of engorgement, corresponds to the formation and existence of the red hepatization. Where cure takes place, resolution follows immediately, and this occurs principally by softening, excretion, and absorption of the croupous masses, and in this process, the alveolar and bronchial epithelium take their appropriate part. The softening occurs through fatty and mucous degeneration of the pus corpuscles, and of the fibrinous substance in which they lie, whereby the corpuscles lose their adhesive properties and become isolated and movable. The alveolar epithelium, which previously undergoes rapid fatty degeneration, is generally changed to granular cells, and hence not only the croupous plugs, but also the cells themselves are thrown off from the alveolar walls to which they at first adhere firmly. We find them often in complete continuous pieces, like a folded hyaline basement-membrane, only distinguishable by the nuclei of the cells composing them and by their surrounding of fat granules. In the bronchial ciliated cells mucous degeneration far outweighs fatty degeneration, so that we may say that the catarrh which has increased to Croup becomes finally catarrh again.* The partially softened croupous cylinders which act as plugs are also lifted off and become movable. The amount of mucus which forms in the smallest bronchi is, as a rule, so great, that the cut surface of the hepatized lung is covered with it and stringy matter remains clinging to the knife blade.

Beneath the alveolar and bronchial epithelium which is thrown off, there begins at once an active cell formation for the purpose of regeneration, recognizable through the ap-

* See also my work on Fibrinous Exudation, Sitzungsberichte d. b. akad. d. Wissenchaft 1863. p. 67. and also Rindfleisch, Lehrbuch der path. Gewebelehre 2. aufl. p. 385.

pearance of numerous nuclei and through young small round cells. I have already remarked that the softened croupous masses were expelled or absorbed. This expulsion is brought about not only by the vital ciliary motion, and the alternate narrowing and widening of the air passages in inspiration and expiration, but also by the powerful elasticity of the lung parenchyma itself. During hepatization, however, the above mentioned factors are, at the outset, abolished and only gradually reappear with the disappearance of the fever and increase of the muscular power of inspiration. As far as the croup extends into the bronchi, so far will mucous degeneration of the epithelium prevail and make the movements of the cilia impossible.

To the same extent, a barrier is also set up against the entrance and exit of air, and to a corresponding degree the bronchi which have a muscular layer are paralyzed, and only follow with difficulty the respiratory movements. The elasticity of the lung-parenchyma, which is especially concerned in expiration, is virtually abolished. Before therefore, the cast-off ciliated cells are renewed, and before the elasticity of the lung tissue and the muscular power of the bronchi have been restored, the expulsion of the fluidified masses is scarcely otherwise possible than by vigorous coughing.

The sputa, which we see in croupous pneumonia, are, therefore, for the most part, catarrhal bronchial products with which the products of bronchial croup are mixed only in those places in which it becomes lost and runs into catarrh. If, then, the alveolar croup spreads but little above the infundibula, every kind of expectoration may be absent during red hepatization, but since it is more often the case that alveolar and bronchiole-croup co-exist, we have the appearance of the well-known fibrinous pneumonic sputa rust-colored by reason of the admixture of red blood globules, which sputa, I repeat, draw their blood and fibrine more from the bronchial mucous membrane than from the lung-parenchyma.

During resolution, the catarrhal bronchial products are of course more abundant, and mixed with them are softened, slightly blood-stained plugs formed in the bronchioles. This proceeds more and more from the bronchi towards the lung-parenchyma, until at last air can penetrate into the latter. An admixture of true alveolar plugs is, indeed, very rare, because of the narrow openings of the infundibula, *the whole hepatized part of the lung retrograding almost solely and alone by absorption of the softened alveolar plugs in situ.* I consider this position important enough to give it especial emphasis, although previously it could not have obtained general consideration.

In order that resolution may proceed in the way mentioned, it is absolutely necessary that the circulation in the lung capillaries should continue steadily and uninterruptedly. Stoppage of blood (stasis) does not belong to an attack of inflammation as we yet hear falsely asserted now and then, in spite of the theory having been long ago disproved, but there occurs only a change in the motion of the blood, which finds its acme in a slowing of the current and always supposes blood in motion. Hence we are always able artificially to make a complete injection of the capillaries of a hepatized lobe. Even during the engorgement the slowing of the current and the increasing compression of the white and red blood-globules reaches the requisite degree. The channel, however, of the retarded stream suffers, by increase of transudation, a diminution in breadth from pressure of the intra-alveolar clot and of the swollen alveolar walls, vessel-walls, and interstitial tissue. Hence it happens that of the forces which in health govern the lung-circulation, (I mean the systole of the right heart and the respiration with its inspiratory aspiration-power and its expiratory pressure,) only the heart systole remains in activity, while respiration fails in the occluded lung. Another point, which may be adduced in passing, viz., that whatever is transuded is taken from the blood, and hence the vessel-walls must contract upon the diminished con-

tents, has little weight, since this situation is shared equally by the vascular system of the whole body, and can have but little influence upon the diseased part of the lung, aside from the fact that the exudation draws from the body chiefly lymph and but little blood.

The relative oligæmia in the hepatized lobe is, however, not without significance for occurrences during the period of resolution; indeed it may be their cause, since it promotes exosmosis (absorption) of the serous infiltration from the alveolar walls, and interstitial spaces, fatty degeneration and softening of the croupous plugs, and mucous degeneration of the epithelium as well as the deposition of fat granules in the lung-framework; indeed it seems as if the prolonged venous condition of the blood in the hepatized parts was even the most essential condition for rapid fatty metamorphosis. All these processes will be temporary if the strength of the patient is not materially diminished by the fever, if the force of the heart,—upon which indeed everything depends,—suffices to drive the blood with greater rapidity through the diseased lung. Not until then can the absorbent activity of the lymphatics again begin, the tissues be freed from œdema and regain their elasticity, the capillaries recover their lost tonicity, and inspiration be able not only to fill the opened infundibula with air, but the capillaries richly anew with blood, and to make the blood-corpuscles accessible to the gaseous interchange of respiration.

So the hyperæmia of engorgement is followed, during hepatization, by an oligæmia which, in resolution, gives place to a new increase of blood until the normal circulation is established. The restitution of the diseased lung is almost always perfect, and it is hardly credible how insignificant a thickening and pigmentation of the parenchyma, how slight an inclination to emphysema in consequence of the elasticity not attaining to its former standard, or how unimportant an adhesion of the pleura may be left behind. If these indications are wanting, every trace of the past is absent. I have

fully convinced myself of this fact by post-mortem examinations after croupous pneumonias which occurred long before and which were thoroughly recognized.

The more significant, then, is the conclusion to be drawn from this, that *croupous pneumonia in fact is only a superficial inflammation*, and that the processes in the alveolar epithelium do not advance deeper into the lymphatics. Lack of physical force and strength of heart's action lead us to expect a fatal issue instead of resolution. The mortality is much increased by the extension of the pneumonia to other lobes, by old age, fatty degeneration of the heart, such as especially occurs in drinkers, in obesity, in weakening from severe diseases, etc., and sometimes there is collateral œdema in other lobes, sometimes intense general cyanosis, and general fatty degeneration (in liver and kidneys). Death at the outset of the inflammation in the red hepatization, is a great rarity. We may examine dozens of those who have died of pneumonia without finding red hepatization, unless one lobe has been affected much later than those first attacked. Death occurs almost always in the stage of the so-called grey or yellow hepatization.

But what is the grey hepatization? The pathologico-anatomical changes are not to be distinguished from those of resolution; it differs only in that it kills, and it kills because the restoring and regenerating activities, by reason of a lowering of the strength, do not overpower the degenerations, or do not keep pace with them, or are perhaps wholly wanting. It differs thus in increase of the degenerations, which experience a significant impulse when the fever and its companion the disease, last beyond the seventh day.

It is called grey or yellow, because the extravasations are blanched, because the fatty degeneration adds its characteristic color, and because the oligæmia continues. There may be at the outset an absolute excess of fibrine and cytoid corpuscles exuded, an exaggerated parenchymatous œdema.

The diseased part of the lung retains its increased volume, weight, and stiffness. The granular dry section of red

hepatization is changed so that the granules (the alveolar croupous-plugs) rise up more readily and are streaked with a clouded stringy mucous fluid. The parenchyma remains empty of air and anæmic, the friability and swelling are unchanged or even increased. The microscope always detects smaller and finer coagula instead of their molecular masses, also mucus, degenerating pus-corpuscles, epithelium, larger epithelial flocculi, and here and there spindle-shaped vessel-cells. In this way croupous pneumonia generally shows itself in the cadaver. During life, the fever symptoms, as already said, continue, instead of undergoing a marked diminution, or they are interrupted by an incomplete remission of some hours only, on the regular seventh day. Although in such a case we expect death as a rule (this occurring often not until the tenth day), yet a delayed resolution may occur; but to attain this result the patient must still retain a moderate amount of his strength. The lung proceeds as from red hepatization and the slower re-convalescence, the more prolonged oligæmic condition and the slight elasticity of the diseased lobe in consequence of the intense parenchymatous œdema are not to be mistaken. A case of croupous pneumonia which came under my notice a short time ago, which died on the eighteenth day of the disease, was a case in point. The parenchyma was variably grey, anæmic and œdematous, the pus-corpuscles in marked fatty degeneration, the epithelium fatty and desquamated, partly regenerated, its cells having multiple nuclei and being in a state of continuous fatty degeneration, while no trace of coagula was to be found.

Such a delay (about to the twelfth day) may cause another serious condition, purulent infiltration. This is indeed to be considered as a high grade of grey hepatization, the coagula have disappeared and given place to fluid, putrid (molecularly destroyed) pus. The most important fact, however, is, that pus-corpuscles are deposited in the lung-framework itself. The pleura shares no less in this purulent infiltration, nor the pleuritic exudation in purulent

contents. If, up to this point, croupous pneumonia is superficial, with a secondary simply œdematous affection of the parenchyma, the purulent infiltration forms a transition to the interstitial forms of inflammation. It is conceivable that if gray hepatization may indeed come often to resolution, this favorable issue is much more seldom with purulent infiltration. When it does occur it happens in complete analogy to grey hepatization with delayed resolution. Swelling and softening of the parenchyma in cases of purulent infiltration must be very great, as can be seen from the fact that the lung breaks easily in removing it, and that a fissure or even a simple finger imprint is quickly covered with the pus which wells up. During life, too, maceration of this or that place may be imminent, and lead to the formation of lung-abscess. It can, however, only come to a complete abscess, when the surroundings and the other diseased parts of the lung give themselves up to resolution and healing.

We see then that at least several weeks are required for the completion of the perfect formation of a lung-abscess, and hence we know why a lung-abscess is such a very rare phenomenon. When the abscess forms, Nature busies herself in limiting it by means of granulations which grow out from the interstitial tissue, and in the most superficial situations, even from the pleura. I will not, however, enter further upon other relations, such as the internal circumscribed adhesion of the lung-pleura and costal pleura, the possible bursting inwardly into a bronchus or outwardly through the thorax-wall, the possibility of its healing after such an evacuation, etc. On the other hand, I must give brief notice to other occurrences which are indicated by authors as the consequences of croupous pneumonia.

Gangrene of the lung, which we know has also other causes (hemorrhagic infarction from thrombosis, embolic infarction, wounds from foreign bodies, etc.), is an occurrence which, although very uncommon, forms usually at the end of the first week of croupous pneumonia, and hence in the stage

of red hepatization. It forms as follows: In some more or less circumscribed vascular territory, in place of the inflammatory blood-movement, there exists actually a stagnation and thrombosis, and hence a condition which ceases to be an inflammation. This is succeeded by a cessation of nutrition in the affected region, and with aid of the entrance of atmospheric air by a degeneration of the whole tissue together with the infiltrated substance to a brownish-black or brownish-green pulpy mass with gangrenous odor and corresponding products of decomposition, with molecular fat, needle-like fat crystals, blood crystals, nucleated pigment-cells with the addition of lowly organized vegetable organisms, etc. The gangrenous spot is not complete, however, until resolution and healing occur in the neighborhood, as in the formation of abscess, and this effects, in a local way, the expulsion or harmless change of the gangrenous masses with limiting suppuration and formation of granulation tissue.

Abscess and gangrene of the lung are two processes which although actiologically antipodally different, yet both lead to the same result, either (and especially with small spots the size of a pea or often larger) to thickening and drying, to gradual enclosing and final cicatrization of the spot, or (particularly with those of larger extent, above that of a hazel-nut, though often less) to loss of substance with excavation, viz. to cavities. Nature succeeds very much more seldom in inducing healing after gangrene than after abscess, on account of the poisonous gangrenous masses being carried into the circulating blood.

Another issue of the croupous pneumonia, which almost all authors endorse, is the development of the so-called cheesy pneumonia. Oppolzer alone * characterizes this acceptation as wrong, and observes that according to his experience, the croupous-pneumonic infiltration did not usually terminate in cheesy metamorphosis. I must express myself with emphasis against the idea that croupous pneumonia ever forms the preliminary stage of cheesy pneumonia,

* See Stofella a. a. o p 562.

and observe, that the opinion was based rather upon theoretical grounds than upon researches upon the living and dead. No one who, at the sick-bed, has treated a croupous pneumonia, and at the post-mortem finds a cheesy pneumonia, is justified in concluding that the former has changed into the latter, but only that his clinical diagnosis has been false. All conditions for cheesy degeneration are wanting in croupous pneumonia, and this holds good especially for the tissue-elements of the lung-framework and its capillaries which always take an essential part in cheesy degeneration. I will adduce the proofs of this position in a more fitting place. I hold the same view also with reference to induration which likewise was characterized as a result of croupous pneumonia. The croupous lung becomes indurated only for the purpose of the encapsulation and cicatrization of an abscess or spot of gangrene, hence only circumscriptly and in special processes, but never diffusely, as marks a lobar spread. Croupous pneumonia is, and remains a superficial inflammation. But I shall return again to this subject.

LECTURE IV.

Desquamative Pneumonia.

Besides catarrhal and croupous pneumonia, there occurs in the lungs a form of inflammation to which my attention was called in 1856,* and which I then described under the name of Desquamative Pneumonia. Since that time I have given to this disease the most careful attention, which seemed all the more necessary, since my own knowledge of it was at that time scanty, and since it was either entirely passed over by other inquirers or confounded with other diseases, being perhaps deemed unworthy of having an especial place among lung-affections, and my investigations have fully convinced me of its importance and great significance. It shall therefore form the central point of my lectures.

I do not speak, of course, of desquamative pneumonia, in all cases where we especially observe desquamation of the alveolar epithelium, for this is a well-known co-existing symptom in most acute lung-diseases, particularly those with marked saturation of the parenchyma, and I have spoken of its occurrence in catarrhal and croupous pneumonia, and in pneumonia from irritation of foreign bodies, as well as in diseases from inhalation of dust. I should consider it as a mistake, if we should give prominence to the desquamation of epithelium in catarrh and croup, for this could lead, and indeed has led to a confounding not only of croupous but of catarrhal pneumonia with the desquamative form. In this way the existence of an actual desquamative pneumonia would be denied, and set aside, and a number of important

* Henle und Pfeufers Journ. f. rat. med. Neue F VIII. p. 57, 62. 80.

sequelæ would be ascribed to catarrhal pneumonia which have nothing to do with it.

A distinction, as sharp as possible, founded upon observation free from prejudice, appears to me to be imperatively necessary.

Desquamative Pneumonia occurs in three degrees and modifications, which should be distinguished from each other. The first kind, the least well marked, and also the lowest in grade, appears as one of the symptoms of severe general processes, and well merits the name of *consecutive* desquamative pneumonia. Since severe general processes are no rarity, it can be examined upon the cadaver more often than the other forms. It has the same significance as the diseases occurring under the same relations in other important organs, such as diseases of the heart, of the muscles, of the liver, of the kidneys, or of the general muscular system, etc., and it can, also, although only very seldom, indicate the principal seat of disease by the manner of its distribution, which is subject to variation whereby this or that organ is more affected than the others.

In the body, the absorption of inorganic poisons, acute exanthematous diseases, Typhoid, Pyæmia, etc., may lay the foundation of this kind of desquamative pneumonia. As is known, the changes in organs occurring in such general processes are called parenchymatous inflammations, hence we speak of parenchymatous myocarditis, parenchymatous nephritis, hepatitis, etc., and by consequence, we must also speak of parenchymatous pneumonia. We shall see that this description is correct, and more than justifiable, yet it is not the one deserving the preference. No one has as yet spoken of parenchymatous pneumonia in connection with the conditions just mentioned, and in short, the participation of the lung in severe general affections, if we except catarrhal and croupous pneumonia, is virtually unknown, for we speak only of hyperæmia, engorgement, hypostasis, œdema, etc., and as yet have sought no more accurate definition of the peculiar process in the lung.

Consecutive desquamative pneumonia appears commonly like desquamative nephritis, on both sides; moreover, being diffuse like the latter, or like parenchymatous nephritis or myocarditis, only of different degree in different places, it is spread over the whole organ, and hence it is lobar. It spreads secondarily to the bronchi (hence it has different relations from the lobular catarrhal pneumonia). In its beginning it may certainly be confined merely to small portions, whereby, especially in reference to the microscopic distinction from hypostatic processes, it is important that we should find it in the front and upper parts of the lung, as well as behind and below. If it is tolerably uniform and clearly marked, the lungs are enlarged, full of blood, with here and there extravasations in spots, either subpleural or in the middle of the parenchyma. The tissue is swollen by serous infiltration, and does not collapse after removal, in spite of the still-existing although diminished amount of contained air, and hardly collapses when cut into. From the cut surface there runs off a finely frothing serum, and soon exceedingly delicate excrescences are formed, which spring from the swollen framework next the empty alveoli. The tissue is wrinkled and easily torn.

These anatomical characteristics, however, are seldom sufficiently pronounced and satisfactory, and microscopical examination is always needed for recognizing the condition with certainty. By this means, we recognize the epithelium in great abundance in the alveoli, the cells are swollen, rounded off, and filled with fine nuclei, which are partly soluble in acetic acid (protein molecules), partly insoluble (fat molecules), the latter soon become predominant. In some cases we can observe the increase of the nuclei, while in others we see sac-like spaces and the exudation of drops of albumen. Free molecules appear through destruction of the cells.

From this process the disease takes its name, the changed cells separate themselves from their bed and from each other, they desquamate, and thereby show the

most complete analogy with the like-named inflammation of the kidney (parenchymatous consecutive nephritis). *As, in the latter, the swelling of the parenchyma, by reason of serous infiltration, gives the reason why the epithelium desquamates*, so also in the lung-inflammation in question. While then, in catarrhal and croupous pneumonia, the principal feature of the process is to be sought in the quantitative and qualitative change of the secretion, on the other hand, in desquamative pneumonia, the main point is the swelling of the stroma which holds the vessels, of the alveolar walls, and of the interstitial tissue, and there is no special secretion.

Here we recognize the difference, as contrasted with catarrh and croup, and know also the reason why the name "parenchymatous pneumonia" is acknowledged as correct. And yet, the name "desquamative pneumonia" is preferable, because the changes in the epithelium can be easily perceived and studied, while the contrary is true of those of the parenchyma. An important peculiarity is this, that *pus corpuscles almost never* come into the field of the microscope, and if they are found in a highly abnormal degree, they are always only carried into the preparation from the concurrent catarrhal bronchiolitis. In like manner, the fibrinous plugs of the vesicles and bronchioles are wanting, as is also, as a rule, any pleurisy.

Just in these particulars lies the distinction from catarrhal and croupous pneumonia. It seems also that, in most cases, the alveolar spaces and bronchioles are, from the outset, so full of desquamated epithelium that the pus corpuscles, produced in consequence of the secondary bronchial catarrh, cannot be driven backwards.

The consecutive desquamative pneumonia may, indeed, in high grades, be a cause of the fatal issue of the general process, which issue occurs through insufficiency of the respiratory organs, and is accompanied by dyspnœa and cyanosis. If the general process does not prove fatal, and if the lung-disease is of the common insignificant degree, it

proceeds to complete recovery, through fatty degeneration of the separated and still later separating epithelium, and through regeneration of the same with concurrent decrease of hyperæmia and swelling, and thus comports itself like the heart, liver, kidneys, etc., in their participation in general processes passing over to cure.

If the general disease does not kill, but the lung is exceptionally severely attacked, so that the processes in it cannot simply retrograde, the affection remains for a longer time before the strength of the patient avails to overcome it. In such a case, we have a *chronic fatty degeneration*, which I have not described partly because the relation of a lung thus affected is already evident from the foregoing remarks, and partly because it is rare in consecutive desquamative pneumonia, but on the contrary, is more common in the second kind of desquamative pneumonia distinguished by greater thickening, where I shall return to it.

Another condition, dependent upon consecutive desquamative pneumonia, which previously was apparently set aside with the title of hypostatic or catarrhal pneumonia, was described by me in 1857* and called acute lung-atrophy. I must ask permission to repeat the essential conditions. "Pleura not adherent, complete absence of air in lung-tissue, unusual stiffness, together with marked saturation, smooth section, brownish-red color, slightly mixed with grey, more or less firm consistence, cylindrical widening and reciprocal approximation of the cartilaginous bronchi with livid coloring of their mucous membrane." Microscopical examination showed besides the want of pus-corpuscles, and fibrinous plugs, that the principal change lay in the lung-epithelium, having indeed the most undeniable analogy to complete acute atrophy of the liver (of Rokitansky). The epithelium was destroyed, the alveoli filled with their molecular remains as well as with a slightly albuminous fluid, and were atelectasic through expulsion of air. Although in no case did a pleural synechia exist, yet the power of

* Virch. archiv. VI. p. 275.

the simple physiological adhesion was so great that it sufficed through the induced diminution of volume of the lung-parenchyma to overcome the walls of the bronchial branches and enlarge their caliber. 'To quote again, "a distinction from acute atrophy of the liver needed only this foundation, that in consequence of the influence of the atmospheric air, the destroyed parts undergo a softening and an acid or putrid decay, and so the pulpy gangrenous spots surrounding the bronchi were established."

In conclusion I will adduce one more especial form of lung-inflammation, and this partly because it appears as *consecutive, i. e.* in consequence of serious positive general diseases, viz. *pyœmic infection*, and partly because its exudations are no less parenchymatous, and follow the track of the lymph-vessels extending in an interlobular and subpleural manner. It has in contrast to desquamative pneumonia the important difference that it goes along with a formation of pus. This is the *purulent interlobular pneumonia* (the dissecting pneumonia of Rindfleisch, lymphangitische pneumonie). Opaque dirty-yellow swelling of the connective tissue-support of the lung, infiltration of pus (*i. e.* abundant exudation of colorless blood-corpuscles, or delayed advance of lymph-corpuscles or rapid destruction of the latter) form the essential anatomical characteristics.

The distinction from other purulent inflammations is simplified, when we consider that the interlobular pneumonia presupposes a centre for pyæmic infection, that it at once gives the impression of being lobar, for the reason that it does not originate and remain in the bronchial-wall, and thence press outward to the parenchyma, but at once, at the outset, establishes itself between the lobules of the latter in lines diffusely obliterated and connected to form a plexus dividing the pleura, in a certain sense, into territories, and also that it deposits no fibrinous coagula or pus in the clear spaces of the alveoli, but only causes a degeneration and desquamation of their epithelium. This process is met with most often, in newly born children which have been infected

with pyæmia from mothers sick with puerpural fever. It occurs, nevertheless, also in adults who die of pyæmia. I have observed it several times after the use of the actual cautery in diseases of the uterus and vagina, and after ostitis of the tibia; indeed, strictly taken, we are to consider as lymphangoitic interlobular pneumonia, every pyæmic pleurisy which does not depend on infarctions from emboli, and in which the layers of the alveolar parenchyma adjoining the pleura take part. I need scarcely mention that death occurs not alone through the local disease, but much more from general causes, and that it is invariable. I may also say that by the same causes there is often produced a mixture of consecutive desquamative pneumonia with the catarrhal or croupous, and in the lower lobes, moreover, the effect of hypostasis is added to all. In the Klinik für Geburtskunde.* I have given an accurate description of this as it appears in the new-born child, and I will briefly repeat it to refresh your memory. "The disease extends generally over whole lobes in both lungs or only in one, it attacks principally one lobe, or develops from the root of the lung backwards and downwards and then forwards and upwards. The unequally infiltrated parts are empty of air, and merge into parts deficient in air and œdematous. The amount of air contained is always very considerable, the color dark-red bordering upon dirty-brown, the lungs feel thick, firm and compact, their tissue is friable, even extraordinarily soft, or mingled with putrefied pus. From the cut surface, wells up an opake black or brownish-red fluid, which, seen with the microscope, consists principally of molecules, nucleated cells and remains of destroyed blood and pus-corpuscles (white blood or lymph-corpuscles). The investing pleura feels sticky by reason of a stringy bloody serous exudation; it is swollen, friable, and easily stripped off. It is, moreover, clouded with a mixture of grey and yellow, and these colors are especially laid on in streaks and meshes, which indicate unmistakably the interstices between the single lobules;

* Hecker and Bühl . B. p. 263.

the surface of the lung, hence, appears lattice-like, divided into polyhedral spaces whose delineation depends on contours and marks, more or less pronounced and principally of a mixed yellowish-grey color. In the situation of the putrid centre, the pleura is often raised up like a bladder. The interlobular tissue is, then, manifestly, the principal seat of the infiltration, which undoubtedly spreads from the mediastinal connective tissue around the thoracic aorta along the intercostal, and especially bronchial arteries towards the root of the lung and between the lobules and lobes of the lungs. The disease therefore merits the name of *acute purulent interlobular pneumonia.* Since a firmer exudation does not exist within the alveoli, it would be wrong to confound the pneumonia proceeding from pyæmic infection with croupous pneumonia. The alveoli are rather simply compressed by the subpleural and interlobular infiltration, and hence become hyperæmic, hemorrhagic, or filled with bloody serum, whereby the epithelium may degenerate and the elastic framework tissue also may undergo putrescent softening."

LECTURE V.

Genuine Desquamative Pneumonia.

After the short digression from my main subject, which the pyæmic interlobular pneumonia occasioned, I return again to desquamative pneumonia, of which I have, as yet, described only the lowest grade, the consecutive form. The second, and indeed most interesting form, and the second grade of the disease, is, in contradistinction to the one we have considered, a primitive inflammation, and deserves the name of *genuine desquamative pneumonia.* It is known by many decisive and well-marked signs. If I may here be allowed to draw a parallel with kidney diseases, I would say that genuine desquamative pneumonia has the same relation to the consecutive form that genuine morbus Brightii has to the affections of the kidney in severe constitutional diseases. No clinical observer and no pathological anatomist will hesitate to concur with me in saying that, in spite of the minute resemblances at the outset when the disease is acute, the two conditions in the kidney should not be hastily pronounced identical. The most severe kidney affection occurring in the course of typhoid fever does not offer (if we except albumen in the urine) in the remotest degree the appearances of morbus Brightii, and although we must confess that general dropsy proceeding from a kidney-change may be observed as a sequel, yet this occurs, perhaps, only once or twice in 300 typhoid cases which end fatally. This uncommon variety is sufficiently significant, and the invasion and progress are quite different

from those of the genuine morbus Brightii, which leads to granulation.

Desquamative pneumonia has a similar relation. It not only bears from the beginning the stamp of an invading lung-disease, but gives rise also to the most important changes in results. It corresponds to genuine morbus Brightii or myocarditis or acute liver-atrophy in being, not merely a concurrent appearance, but the localized expression in the lungs of a general disease.

In the study of this affection, we are generally confined to an advanced stage (after 4 or 6 weeks' duration or even more), since the disease does not seem to prove fatal at the commencement. At this time, however, its signs are so clear and well-marked that I always wonder why other observers have as yet taken no notice of it.

The anatomical signs of genuine desquamative pneumonia are much more characteristic than those of the consecutive form. It may, indeed, be limited to one lobe of one lung, and in this case it is commonly the upper lobe, or it may attack one entire lung, which is much more common, and then the upper lobe is usually more deeply affected than the lower, or still again, it may attack both lungs either only in the upper lobes or in all the lobes, and in this case the disease in one lung is, as a rule, more severe than in the other. *Almost always the process in the upper parts is most developed and its progress from above downwards evident*, although in very acute cases, all portions seem attacked simultaneously. The same grade and the same age of the disease is, therefore, seldom found in the different lobes. In cases where the disease is 6 or 8 weeks old we find the volume and weight of the affected lobe or lung significantly increased, the surface smooth, faintly glistening, covered with fibrillated fibrine or adherent by reason of organized connective tissue, the pleura itself being swollen and here and there beset with ecchymoses especially upon the lower lobes. The parenchyma does not collapse either in the removal of the lung from the thorax, nor after

section; its elasticity seems limited or abolished, and the tissue remains rigid. Yet its friability is much increased, and augments with the duration of the disease. The cut surface exhibits in a marked manner the lobar, diffuse spread of the affection, while the air-contents are more or less diminished or in places even abolished, and a weak granulation is seen, which is, however, the reverse of that in croupous pneumonia, for while the granulations in croup are formed by the fibrinous plugs which project from the alveolar spaces, or indeed, in a certain degree are pressed out (since the cut œdematous interalveolar tissue gives out water and its elastic fibers retract), here, on the contrary, the thickened interalveolar parenchymatous parts, rigid by reason of swelling and adherent infiltration, form the granular prominences between the alveoli which are opened and emptied by the stroke of the knife. The amount of blood which the lungs contain is very considerable, and the longer the process has lasted the greater is the amount of granular pigment deposited, hence the color of the tissue is greyish or even black. In many cases we find the friability so marked that it constitutes actual diffuse softening, which may embrace the greater part of a lobe. In spite of the manifest swelling only a scanty gelatinous fluid can be obtained by scraping the cut surface (because the infiltration, as described, seems firmly adherent to the tissue), and this fluid is bloody and opaque—through the admixture of countless cell-elements.

This infiltration forms the principal difference between this affection and the consecutive desquamative pneumonia, which shows only a saturation with serum, while the exudation of the genuine desquamative pneumonia is of a plastic productive nature. With the microscope we find almost nothing but cast-off epithelium, belonging not only to the alveoli, but also to the finer bronchi, filling these spaces, as already said, often to the exclusion of air. The epithelium is in a state of marked fatty degeneration, *i. e.* the cells are large and distended with a mass of fat molecules which

conceal the nucleus, in other words they are changed into granular cells, and many of these contain brown or black granular pigment. *Pus-corpuscles, mucus, or coagula are wholly wanting.* This fatty degeneration may be traced far into the bronchi, even as far as the large primary divisions. The more extended this is, so much the more uncomplicated is the desquamative pneumonia, and so much the less does catarrh in the bronchi coexist, *i. e.* the abundant muco-purulent secretion, characteristic of catarrh, is more or less wanting, and in its place is found a gelatinous, albuminous fluid, in which the cast-off fatty, ciliated epithelium is suspended.

In more recent spots, we see rather the beginnings of the changes described; in particular we find the scarcity of air, the friability, the pigmentation, and the fatty degeneration less marked, but the blood contained in the lung is, on the contrary, present in greater quantity; softening and atelectasis are of course not to be thought of in this stage.

These conditions are of the utmost clinical importance, for they give the *sputa* a definite unmistakable character. Even in the first week, in which this disease might be confounded with croupous pneumonia by reason of the fever, the general crepitant rate, a percussion-note somewhat resonant, often clearly tympanitic, mixed or even bronchial breathing and bloody sputa, sure indications may be obtained by microscopical examination of the sputa, signs that have nothing to do either with catarrhal or croupous pneumonia, but only with the desquamative form, for neither in catarrhal nor croupous pneumonia does the alveola-epithelium occur in such quantity in the sputa, having ciliated epithelium mixed with it. The longer the disease lasts the more abundant are the epithelial and granular cells, the more advanced the fatty degeneration, and the more pronounced the cell-pigmentation. Free fat molecules, free cell nuclei, proceeding from breaking down, are, of course, not wanting.

In course of time another remarkable degeneration

appears, the myeline, which likewise is seated in the alveolar epithelium. The enlarged, rounded-off epithelium cells become in like manner granular, but they have quite a different appearance from the fatty granular cells, for the latter, under a low power, give the impression of dark black bodies by reason of the broad shadow of the outlines of their molecules which only glisten strongly in their central points, while the myeline, on the contrary, glisten fully, their outlines are faint and difficult to see, and hence they make the cell appear as extremely pale, clear corpuscles.

From the mass of myeline cells and of free myeline in the sputa, we can approximately decide upon the greater or less duration of the process which stands in direct relation with it, if we perchance have not observed the patient from the outset and the history be deficient.

The lung-epithelium shows one more characteristic, viz. a proliferation of the nuclei (not as in croup, endogenous free formation of pus corpuscles), for we perceive younger forms indicated by the different size and figure, mere elementary bodies, which we may consider as indicative of regeneration, even *exuberant regeneration.* Desquamative pneumonia, even at the outset, is characterized by this plastic activity, while in croup regeneration indicates the final healing act of the process. Among the forms of regeneration are also to be reckoned the stellate and spindle-shaped cells of various development and formation, which we can get by scraping the cut surface. They, like the epithelium, contain here and there pigment granules. The principal mass of the black pigment often is so abundant that we are led to think of "siderosis" (deposition of peroxyd of iron), or of "anthrakosis," which latter may be rejected by reason of the simple molecular form and size of the pigment granules, by reason of the chemical peculiarities, and in somewhat larger fragments, by the absence of those botanical signs which belong to carbonized plants, especially to their woody parts. The pigment is seated in

molecular form in the succulent lung-tissue itself, and we can discover deposited here and there upon the walls of the finer blood-vessels, brown or black pigment granules. The framework, moreover, is thickened by new layers of connective tissue, as is indicated by the stellate and spindle-shaped cells, and hence we see in the larger connective-tissue bands an alternate streaking of white and black, for the thickened peribronchial tissue is distinguished by a white color.

These relations (rigidity of the parenchyma, pigmentation, proliferation of the connective-tissue and of the epithelium) point much more distinctly to parenchymatous processes than is the case in the consecutive desquamative pneumonia, and the genuine desquamative pneumonia merits, indeed, much more than the latter, the name of parenchymatous inflammation. Yet it seems to me more fitting to retain the appellation desquamative, since at the bedside the diagnosis of the desquamative form, like that of morbus Brightii, is simply and only to be fixed by the microscope. The blood-vessels are, as a rule, somewhat dilated, and this capillary ectasis, the pigment formation, and the thickening show at first sight an analogy to brown induration. But these two diseases can scarcely be confounded, since in the former the heart disease which produces the condition is wanting, and the sudden inflammatory outbreak and course, the black (instead of reddish-brown) pigmentation, the excessive fatty degeneration, and the myeline degeneration of the alveolar epithelium aid in making the distinction, and in especial the peribronchial thickening with its white color, together with the capillary ectasis limited to the section where thickening has not occurred and of only moderate degree, offer sufficient grounds for diagnosis. Marked dysprœa, cyanosis, and cough accompany the disease in question, and secondary fatty degeneration of almost all the organs of the body, especially of the heart, combined with dilatation of this organ, lead to a fatal termination. I have previously * sought to refer

* Virch. Arch. 1859.

Rokitansky's hypertrophy of the lung to brown induration; perhaps the same observer has also classified under that head genuine desquamative pneumonia which has passed into the chronic stage.

Physicians have, up to the present time, comprehended these cases at the bedside according to their stand-point in a variable though scarcely correct manner. Unfortunately there are only a few in whom the results of post-mortems have produced the conviction that their views need reforming.

In the consecutive desquamative pneumonia, I omitted to speak of the alveolar epithelium as endothelium, for in that connection the subject possessed only a subordinate interest. It were more to the purpose to consider it in connection with pyæmic interlobular pneumonia, but the seat of pyæmic disease is rather the interstitial connective tissue, not the alveolar parenchyma, at least not from the outset. It is not until we reach the genuine desquamative pneumonia that the relations and the analogies of the connective tissue corpuscles, lymphatic-endothelium and alveolar epithelium come forward so prominently as to show that the desquamation of the latter is due not only to the swelling of the alveolar wall, but must be regarded as the proliferation and degeneration of similar tissue elements. Although I have up to this time described desquamative pneumonia as an acute inflammation leading to a fatal issue, it is not my meaning that it always proves fatal. On the contrary, from observations at the bedside, where, besides the peculiar course, and the physical symptoms, the microscopical examination of the sputa established the diagnosis beyond a doubt, I have been convinced that this, like genuine morbus Brightii—and perhaps quite as seldom—may pass over to *complete recovery*. It is more often the case that, where death does not occur, lasting and irremediable changes are induced either by a progress slow in development, or one which advances faster but has frequent relapses; changes which, although they develop out of

one and the same fundamental process and hence are intermingled, yet require an especial study. Here belongs, in the first place, *chronic fatty degeneration*, of which I have already spoken in connection with consecutive desquamative pneumonia.

This is not, however, only a form of the previously described genuine desquamative pneumonia prolonged much beyond the time during which we may speak of an acute or sub-acute process, but it is distinguished by special changes, from this form and from the continuous forms of desquamative pneumonia. I have remarked cases of chronic fatty degeneration spread over both lungs which could be proven to have *lasted a whole year before death occurred* under the clinical appearances of phthisis. If the desquamative inflammation is limited to one lung, or only one lobe, or a part of a lobe—and for the two last-named varieties, as a rule, these are the upper lobes or their upper portions—the chronic fatty degeneration remains limited. It also becomes circumscribed where the inflammation in one part completely recedes, but in another (especially that situated toward the apices) opposes reparation, and falls into a chronic fatty degeneration. As we might expect *a priori*, with longer duration, the signs of general hydræmia appear, which are reflected in the diseased lung by diminution of the capillary ectasis, and as a result, the lung affected with chronic fatty degeneration is characterized by pallor. In spite of this, the volume and weight diminish but little, while, on the other hand, the pigmentation diminishes so much more in consequence of absorption and saturation, that the color becomes light slate-grey, and we see in this somewhat darkened ground, whitish points which appear like sprinkled meal. These whitish points are nothing but alveolar spaces which are filled with masses of epithelium in fatty degeneration. The quantity of air contained is small, not for this reason alone, but also because of the constant and considerable swelling of the stroma (Rindfleisch's inveterate œdema), which although

somewhat thicker and friable, yet remains inelastic and does not collapse. As in the epithelium, we see here and there also a fatty degeneration belonging to the spindle-shaped cells of the parenchyma, while myeline degeneration in the epithelium is likewise more or less represented. Chronic fatty degeneration might be confounded with a subsequent stage of croupous pneumonia through external similarity, but the microscopical investigation (which in croupous pneumonia always reveals pus corpuscles, however changed they may be), the scarcity of pigment, and the thickening decide in doubtful cases.

LECTURE VI.

Cirrhosis of the Lung.

ALTHOUGH up to the present time desquamative pneumonia has not been awarded its due importance in the literature of this subject, we must not think that the results which I have given concerning this affection have escaped investigators; for this is not the case. All who have worked industriously upon pathological histology, and have made the lungs the field of their researches (Virchow, Colberg, E. Wagner, Rindfleisch, etc.), describe these results often in detail, and even use in their description the phrase "desquamation of the epithelium of the alveoli," but always under the heading "catarrhal pneumonia." Although I was, perhaps, the first to observe the facts bearing upon this subject, I have to-day nothing absolutely new to communicate. On the other hand, I shall try to show *that its present pathological significance as catarrhal pneumonia is not correct, and shall seek to bring forward a new conception; to establish genuine desquamative pneumonia as a distinct disease, having several grades, and shall charge it with being the preliminary stage and companion of significant, dangerous processes, lung-phthisis and tuberculosis, by bringing it into genetic connection with these conditions, which are already more or less understood.*

In my previous lectures I have already brought before you one result of genuine desquamative pneumonia, viz. chronic fatty degeneration, which can only end fatally. My present letter concerns a second result, the LUNG-CIRRHOSIS of Corrigan. I have already shown that the swelling

of the lung-framework, of the alveolar walls, and of the interlobular and interstitial connective-tissue bands lays the foundation of desquamative pneumonia, and that the desquamation directly succeeds the swelling. I have also shown that the essential difference between the consecutive and genuine desquamative pneumonia lay in this, that, in the former, the parenchymatous swelling was occasioned by a serous exudation, in the latter by one of a more plastic nature, characterized by proliferation of the alveolar epithelium and of the connective-tissue corpuscles. Now, in cirrhosis the latter condition (I refer to the growth of the connective tissue elements) obtrudes itself so prominently that the superficial epithelial changes almost escape notice. Under the microscopical conditions of genuine desquamative pneumonia I especially mentioned that, in the acute stage, we may, by scraping the cut surface, obtain from the compact jelly-like infiltration of the stroma, spindle-shaped and stellate cells, the diagnostic signs of embryonal new formations of connective tissue. The latter leads in chronic course to *connective-tissue hypertrophy* (cirrhosis), and the common name "*chronic interstitial pneumonia*" means, in fact, nothing more than this issue of genuine desquamative pneumonia. To use again my comparison, cirrhosis is to desquamative pneumonia, what granular kidneys are to genuine morbus Brightii, what granular liver is to parenchymatous hepatitis, what induration of the heart is to parenchymatous myocarditis. Although the process begins very early with the insignificant thickenings mentioned in the former lecture, and with simultaneous diminution of the volume of the lung, yet we do not use the name cirrhosis until the connective tissue is formed anew, so to speak, in masses, when it appears like *a fibrous scar or tumor by which the alveolar parenchyma and the finest bronchi are enclosed, obliterated, and destroyed.* Among the spindle cells are often found *muscle cells;* indeed, frequently the whole hypertrophy seems to be of a muscular nature, and to me this last serves as an important means of proof

of the existence of muscle cells in normal alveolar parenchyma. (Moleschott, Piso-Borme.) For these cases the term *muscular-cirrhosis* would be appropriate, to distinguish them from the ordinary *fibrous cirrhosis.* Fibrous connective tissue is thick and hard, dry, poorly furnished with vessels and blood, and we might expect that, in the lungs, as elsewhere, it would have a white color. But the lung has a priority in pigment formation, and in cirrhosis a strikingly large amount of pigment is deposited (hence called *slaty induration*, by Rindfleisch). We find a blackish coloring in the place of the former alveolar parenchyma, and this alternates only with straight or branched white, pigmentless, fibrinous bands, or, studied in cross-section, with white points and small stars, which represent, not the alveolar tissue, but the sheaths of the terminal bronchial prolongations (*peribronchitis fibrosa*). The tendency to pigment formation occurs in the lungs, possibly also upon chemical grounds, but especially because the respiratory capillary plexus in all hyperæmias and inflammatory conditions becomes readily dilated and so gives occasion to the transit of blood globules, which at first are only taken up by the epithelium, but afterwards are carried farther on in the lymph passages and juice canals (saft-kanälen) and finally come to rest.

Desquamative pneumonia is, therefore, the cause not only of the thickening, but also of the black pigmentation, and the latter is one of the main characteristics of cirrhosis. There is only one especial form of tissue thickening which is colorless, or much oftener *transparent grey*, and which is seldom found alone, but occurs in company with fibrous black cirrhosis, viz. *amyloid cirrhosis* (the third form). The capillaries and finest arterial twigs, and especially the encircling vessels of the alveoli, are glassy and gelatinously swollen, and the interlobular connective tissue is thickened in streaks through cellular growth. The amyloid envelope of the vessel-wall is a hindrance to the exit of red corpuscles, and consequently to pigmentation. An entire section

of the lung-tissue may show this peculiarity, often forming a confluent, nodular mass, often a radiating interrupted plexus, often following the finest vessels demonstrable only with the microscope, and the condition is evidently the analogue of the amyloid kidney, the amyloid liver, etc.

I have often asked myself whether amyloid degeneration in the lung has the same general relations as elsewhere, especially whether it indicates that *syphilis* has affected the general constitution; but the number of cases I have examined with reference to this point is too small to enable me to give a satisfactory answer. Yet it is worthy of mention that half of these show acquired syphilis; whether the other half belongs to inherited syphilis, I cannot decide. I recommend this subject to your investigation.

Cirrhosis shows better than any other change how desquamative pneumonia diffuses itself and spreads downwards from the apex of the lung. In the commencing stages this condition is but slightly pronounced, and the lung appears to undergo in the whole circumference of the inflammation a more or less slight degree of thickening, as we see it, for instance, in chronic fatty degeneration. Soon, however, the increase of stroma in the apex surpasses the rest and progresses without interruption to the lower parts, keeping equal pace with the pigmentation.

Since desquamative pneumonia has relapses, and since it often extends insidiously over broader districts of the lungs, not at once from the outset, but only after several relapses and returns, the difference in development between the changes in the apex and those in the lower parts may appear prominent. Indeed, we often find, separated from the cirrhotic main body, in the lung-apex and below this, *fibrous black nodules* from the size of a hen's egg to that of a hazel-nut, distributed singly or in groups. After the extension and the retrogression of the inflammation it may also happen that cirrhosis invades only the lung-apex, or perhaps one-third or one-half of the upper lobe, or the whole upper lobe, or in some cases even a part of the lower

lobe also. Single small nodules in the lung-apex have an especial interest, because they are common and appear as nature's cure. According to statistics 30.4 per cent. of those affected with cirrhosis have *apex-cirrhosis* alone, while those who die in the chronic stages of desquamative pneumonia give 43¼ per cent.

Although the boundaries of the black nodules seem sharply defined, yet the transition to the remaining lung-tissue, as we find when we look closely, is not abrupt, but they stand connected to the latter by means of connective-tissue elements which merge into both. I cannot here forbear mentioning one interesting though very rare occurrence. The cirrhotic nodules may often *ossify* and betray this change upon section. Such nodules appear finely porous, and have a rather whitish-grey color with pigment sprinkled in. The bony substance follows the fibrous bands, permeates the nodules in undetermined points, lines and branches in all directions, and contains true osseous corpuscles. Between these there is degenerate fatty, or even chalky epithelium, which gives rise to the greyish-white appearance. Virchow has treated of them in his work on Tumors.* They deserve the name "*diffuse lung osteoma*," to distinguish them from the *branching* variety which only correspond to broken-off interstitial connective-tissue hypertrophies.† As everywhere in the body where embryonal connective tissue finally passes over to completion, the broad capillaries, newly formed in the stage of the first development, become narrower and narrower, and many of them disappear; and even more marked is the destruction of the original normal vessels; indeed, one may say that often the whole previous respiratory network perishes, and in its place we see a scanty network just as in fibrous tissues. Coincidently with the loss of the alveolar epithelium, the beginnings of the lymphatics experience a like destruction. This is the same procedure as in granular

* II. Bd. p. 103.

† See my article in Sitzungsberichten d. b. Akad. d. Wiss. 1867.

kidneys or in granular liver, in which the capillary plexus, established for the secretion of urine or bile, perishes. In comparison with this the brown induration has quite another relation, for in that case there is rather a capillary extension and dilatation of the respiratory plexus.

The more remote consequences, too, of the abolition of the capillaries are full of significance. Provided, namely, that the general amount of blood is scarcely lessened, and also that the blood in the right heart, belonging to, and destined for the lungs, is not essentially diminished in quantity, then, in proportion to the lessening of the channels of the blood in the short circulation, the hæmostatic pressure must become correspondingly increased in a backward direction, *i. e.* in the pulmonary arteries, in the right heart, and in the general venous system, and give rise to dilatation. The blood itself, too, becomes insufficiently subjected to gaseous interchange—and heart symptoms, cyanosis, nutmeg liver, congested kidneys, dropsy, etc., occur speedily.

Since the lung-capillaries receive blood from two sources, the increased blood-pressure will make itself felt also in the *bronchial arteries* and produce a secondary catarrhal bronchitis of the larger bronchial branches.

The foregoing explanation is important as enabling us to deny decisively that the cause of all these conditions, especially of cirrhosis, is a common catarrhal bronchitis. It would be a grave error to maintain that the development and formation of these processes, instead of being from the lung tissue toward the bronchi, were rather from the bronchial mucous membrane toward the lung-parenchyma. The fatty and myeline degeneration of the alveolar and bronchial epithelium, which characterizes desquamative pneumonia, is somewhat changed in progressive cirrhosis. On the one hand, less and less of the epithelial forms and of their nuclei are seen, and finally there remain only remnants composed of fat molecules which change the black color of cirrhosis to a light slate-grey. The fat

molecules, which are grouped in little heaps, correspond clearly to the former alveolar spaces, but often they are arranged in streaks and represent the walls of the former vessels and the connective-tissue corpuscles or the muscle-cells of the alveoli. On the other hand, the myeline degeneration may be very excessive, and I have been able to remove from enlarged lobules and bronchioles collections of myeline visible to the naked eye. These cases coincide with the observations of Friedrich,* and are of the same nature, where in cirrhotic and brown-indurated lung-tissue, besides the plugging of the finer bronchi with a yellow mass, there were found lamellated bodies (corpora amylacea) of .02‴ to .12‴ in diameter, whose jelly-like substance, colored in the centre and circumference by crystalline pigment, was split concentrically and radially.

It is conceivable that, in the alveolar and bronchial obliteration, *secretions* in the branches which lead immediately from the obliterated bronchi remain at rest, become thick, *cheesy* and *chalky*, and so much the more if secondary bronchitis exists. This condition, limited to a small region of such branching bronchial tubes, borders upon the so-called *chronic fibrinous bronchitis*, in which the branched clot may be coughed out. Sections of cirrhotic lung tissue may therefore show, besides the previously described black and white color of the fibrous formation, also the dusty greyish-yellow of fatty degeneration, the greyish jelly-like appearance of myeline and amyloid degeneration, the yellowish-white of the cheesy centres, and the chalky-white of those which are petrified, in short, *an appearance like granite.*

The smaller circumscribed cirrhotic nodules pass over anatomically in a continuous series into the peribronchitis, while ætiologically the diffuse cirrhosis dependent upon general causes merges with the *cicatrization and encapsulation* of reaction against local lesions (from foreign bodies, catarrhal pneumonia, etc.). A scientific line of distinction is *in concreto* impossible.

* Virch. Arch. Bd. IX. and X.

I have now two more occurrences to bring forward, which must not be forgotten in mentioning the characteristics of cirrhosis, viz. *hypertrophic bronchiectasis and pleuritis.* In speaking of the bronchi I have as yet described only the obliteration, the destruction of the finest terminal ramifications, the secondary catarrh, and retention of its products. In the spots where the compact cirrhotic masses do not develop, bronchiectasis may arise. Connective tissue just formed through inflammatory processes, always acquires through its succulence greater volume than the completed tissue which results from it, but its completion is marked by contraction. However marked the embryonal new formation of connective tissue may have been, and however much the volume of the diseased portion of lung may have increased, there appears with the formation of cirrhosis, a *lessening of diameter in all directions*, and on the one side, a concentrically acting force will be exerted upon the chest-walls, while upon the other an eccentrically acting traction is brought to bear upon favorably situated, unobliterated bronchi. The cartilaginous bronchi will especially feel this traction and become enlarged, and since their walls in the present case are directly surrounded by cirrhotic lung-tissue, the term "*hypertrophic bronchiectasis*" has become necessary to distinguish it from the "atrophic" previously described.

What I observed concerning retention of secretions applies in full force to enlarged bronchi. On the part of the alveoli and bronchi the elastic and contractile force no longer exists to remove the secretion, the *vis a tergo* is wanting, and only where the walls are not thoroughly rigid can a portion of the secretion be thrown out by a powerful compression of the lungs from abdominal pressure, comparable to an expiratory emesis. I need scarcely say that the entrance of air induces disorganization and putrescence of the secretion. But the *chemico-mechanical action* of the disorganized secretion has great influence upon the mucous membrane of a dilated bronchus. Soon the epithelium is

destroyed, the mucous membrane sloughs and is cast off by a line of limiting suppuration, the cirrhotic lung-tissue laid bare by this process lies close to the putrescent substances; then follow new sloughing, new removal—this time from the lung-tissue itself; in short, the hypertrophic bronchiectasis has become a *chronic bronchial cavity*, with cirrhotic walls, here lined with sloughs, there with fatty tissue, here with granulations and pus, there with a smooth fibrinous layer, and only in a few places are found islands of sound mucous membrane with ciliated epithelium upon them. This *secondary* formation of cavities, occurring in cirrhosis, is to be distinguished from other methods of origination of cavities.

Cavities like these, which are small and shut off, contain cheesy and myeline masses here and there mingled with cholesterine. In larger ones with free communication with the exterior, are found greyish-yellow putrescent masses of molecularly disintegrated pus, sloughy and cheesy parts of the alveoli. (Cavernen bröckchen, Schroeder, v. d. Kolk), and blood is often mixed with it, sometimes almost clear, pure blood, while in still larger ones the retained contents are changed to an inodorous or fetid greasy substance of a grey color, which consists of detritus, margerine needles, and cholesterine.

The pleura has an equal interest. The interstitial connective-tissue tracts of the lung parenchyma run out under the pleura, and it is easy to understand why the pleura shares in the swelling and new formation of connective tissue. Although this participation can be recognized already in the acute stage of genuine desquamative pneumonia by means of jelly-like swelling and opalescence, loss of lustre, velvet-like softness of the serous surface, the latter being caused by change of epithelium (enlargement of the cells to immense dimensions with corresponding proliferation of large nuclei), yet in the stage of cirrhosis it appears differently. The epithelium undergoes fatty degeneration, and desquamates like alveolar epithelium, and

beneath it develop villous connective-tissue formations containing young embryonic capillaries (*desmoid fibrine.**) Soon there occurs an abundant *transudation* of serum (sero-fibrinous exudation), either clear or mixed with blood from the friable and ruptured new capillaries (hemorrhagic exudation). This blood flows over the vegetations, and compresses the lung, but soon after, in most cases, the affection ends with fibrous *adhesions* of single lobes to each other, and of the surface of the lungs to the costal surface. Since cirrhosis shows its greatest intensity and spread in the apices, and decreases from that point downwards, we find the pleural connective-tissue hypertrophy (the desmoid fibrine, the skin-like, callous adhesions) also the most marked over the apices of the lungs, and decreasing gradually as we go downwards. Although at the apices the thickness of the adhesions may exceed 1 c. m., at the lower lobes it often appears more like a spider's web or is wholly wanting. It is evident that the adhesions may be enlarged, not only through their own growth, but also through cirrhotic lung-contraction. The same traction which leads to bronchiectasis is effective here, since the capacity of the thorax will not permit of further contraction. (I refer to the traction by shrinkage, evidently acting from the surface in radiating lines towards the root of the lung.) The space, thus made, is filled out by a thickening of the pleura. The pleural desmoid formation is important, especially in the region of the hilus, since there the larger vessels and bronchi are ligated; and on the one hand stasis, hemorrhage, and thromboses are produced, and on the other, dyspnœa with fresh cause for bronchiectasis. The statistics of older changes in the cadaver which my former assistant *Bollinger*, now Professor in Zürich, undertook at my suggestion,† support, statistically, the frequency of pleural growths, and especially of those connected with condensations of the lungs. It appears that three-fourths (73¼ per cent.) of the cadavers,

* Vid. Sitz-Ber. der b. Akad. der Wiss. 1863.

† Deutsch. Archiv. f. Klin. Med. V. p. 142.

more of the male than of the female sex (76.4 per cent. to 68.2 per cent.), were affected with pleuritic adhesions. By far the greater number were on both sides (62.2 per cent. to 13.5 per cent.), showing the common origin of the disease. Especially close adhesions were found in the upper lobes in 26 per cent., of which 33 per cent. were referred to cirrhosis, while, on the other hand, adhesion was general and complete only in 14 per cent.; and in 66 per cent. cirrhotic thickening of the lung was present. The statistics of the adhesions on the right side preponderated, both as to extent and degree. It is easy to avoid confounding the layer of pleuritic effusion occurring during the progress of cirrhosis, with a primary chronic compressing pleurisy. Aside from the situation (the compression from pleuritic exudation being in the lower lobe, that from cirrhosis in the upper) the whole nature is different. Cirrhotic tissue loses relatively little space, and consists essentially of fibrous black tissue. Compressed lung-tissue is, on the contrary, markedly reduced, simply thrown out of use, the elastic arches of the parenchyma are irregularly twisted together, and there is almost no hypertrophy of connective tissue. In cirrhosis, bronchiectasis is not necessary and essential, and if it occurs, it attacks generally one or two bronchial branches, is of the sac-like form and often ulcerated. In compression, bronchiectasis is essential, regularly distributed and always cylindrical, and the mucous membrane is intact. From these points of view should be considered also a chance concurrence, viz. the compression of cirrhotic lung.

LECTURE VII.

Cheesy Pneumonia.

With chronic fatty degeneration and cirrhosis I connect *cheesy pneumonia.* This develops, like those processes, only from pronounced genuine desquamative pneumonia, and is its highest grade. Those forms of genuine desquamative pneumonia which show no cheesy change I will henceforth always call "*pure genuine desquamative pneumonia,*" for the sake of distinction. Indeed, we may, in very rare cases, see the consecutive desquamative pneumonia also become cheesy, and I insist that without microscopical examination it is almost impossible to decide whether the yellow color comes from the pus of catarrhal pneumonia or of purulent peribronchitis (of which more in the next lecture), or from its cheesy walls. I will also call attention to one more point of differential diagnosis, since it has already happened, at the post-mortem table, that cheesy pneumonia has been confounded with cancer of the lung. It is self-evident, that by this cannot be meant the secondary medullary cancer appearing in nodules, but the primary cylindrical-epithelium cancer (or rather, the papillary and acinous epithelioma of the lungs*), which in fact has at first sight a deceptive simi-

* One who has opportunity to examine such a primary epithelioma will soon be convinced how entirely different the formations springing from true epithelioma are from the well-known endothelium-like growths which arise from alveolar epithelium. . The epithelioma develops on the ends of the bronchi in form of hollow sprouts of cylinder-epithelium destitute of cilia, often clearly lamellated, which grow into the lung parenchyma and partly compress it, partly cause absorption, and partly pass directly into the alveolar spaces, enlarge

larity to cheesy pneumonia, and is only distinguished from this disease by a white instead of a yellow color. The microscope removes all doubt at once. Moreover, the diffuse cancer-infiltration that, in careful examination, is found to follow the lymphatic plexuses of the lung-parenchyma, attracting attention at once by its white color, and still more by the numerous subpleural ramifications, has perhaps already been mistaken for cheesy pneumonia. Finally, we must mention the lymphatic pyæmic interlobular pneumonia, which I have already in my second letter sufficiently characterized—for a diagnostic error seems to me credible.

You know that by the word *cheesy* is indicated the product of a degeneration which, under certain circumstances, may take place in any tissue, normal as well as pathological. By "*cheesy pneumonia*" we may therefore, in effect, understand, not an especial form of inflammation of the lung, but only a pneumonia which is inclined to fall easily into cheesy degeneration. Such a pneumonia is the degenerative. With reference to the demonstrable changes the cheesy degeneration is indeed nothing but a fatty degeneration. It must, however, be distinguished from this, because it is marked by absolute anæmia and continual absorption of water, hence by pallor and dryness; while the conception of true fatty degeneration requires a constant flow of blood through the part and an increased amount of tissue-moisture.

Every inflammation goes over finally into fatty degeneration, for, in every inflammation, it being a vital process, activity of circulation and swelling are essential signs, and the fatty degeneration is only the result of the mal-nutrition coming from inflammation, or, partly, of the endeavors to bring back into a normal condition the changed and

correspondingly, and cause the latter to have the deceptive appearance of having their walls lined with cylinder-epithelium. If the infundibula were in the first place lined with epithelial acinous formations, the point of origin for the epithelioma would not be originally the ends of the bronchi, but the expansions of the infundibula.

disturbed circulation and nutrition. Cheesy degeneration, however, is a process in what is already dead, and has quite another preliminary, viz. *sloughing.* This exists everywhere, where a capillary region has been, from some cause, completely cut off from its stream of blood in a rapid or sudden manner, so that the part, of necessity, perishes. Sloughing may occur together with inflammation, indeed, in the inflammatory region itself, yet it is no part of the inflammation, but rather a cessation of the latter—its opposite —it is death (anæmic necrosis). *It is, therefore, the necrosed tissue which undergoes cheesy degeneration;* yet in this connection we must notice that it remains in contact with the living tissue full of circulating blood, and through osmotic interchange undergoes changes which consist in fatty degeneration and absorption of water. Although itself inactive, it is not shut off from the nutritive activities of its border regions, and may, in proportion to the smallness of its circumference, be absorbed even to the last molecule, and disappear.

If the sloughy tissue is already decayed and appears pale or yellow, these peculiarities are more pronounced in cheesy degeneration, and the tissue is changed into a material comparable to yellow friable cheese. Considered as to time, the sloughing is somewhat acute, belonging to the acute stage of a process; for cheesy degeneration a longer duration must be allowed, and this appears always somewhat chronic. Wherever a portion of tissue dies, bacteria swarm, whose germs are always present in our bodies, however secluded from the outside the part may be. They may be found, *e. g.*, in every thrombus, not only in those resulting from puerperal and pyæmic processes, but also in every thrombus-like deposit of a genuine endocarditis; indeed, they even appear in every part where there is considerable slackening of the current of the blood and tissue juices: *e. g.* in inflammatory patches, and in regions of venous congestion. Hence we do not wonder when we find them in cheesy masses, as V. Recklinghausen has already mentioned. They appear

generally as bullet-like bacteria (microeveci) and as short jointed rods.

If we apply these general considerations to cheesy pneumonia, the processes which they call into existence will be clear. We must distinguish two stages, first, an acute—and then a subacute and chronic stage.

A. *The acute stage.*

In this stage a lung appears as follows:—It is enlarged in volume and weight, has upon section a striking resemblance to red porphyry, since there appear, deposited upon a red ground, whitish-yellow, large, small, and very small granules, isolated and aggregated. The *red ground* owes its color to the abundance of blood, and is generally dark red, less often clear flesh red. The air contained is small in quantity or entirely wanting, and although the tissue is considerably swollen and friable, yet it sinks beneath the level of the cut surface of the whitish-yellow parts, and we can, by scraping, obtain a noticeable amount of a transparent jelly-like fluid (jelly-like infiltration, gelatinous infiltration of Laennec), in which we can see, besides red blood corpuscles, larger spherical or flattened, sharply outlined, strongly refractive, transparent cells, here and there yellowish brown, or grey by reason of fine granular contents, and holding one or more nuclei (the gelatinously swollen desquamated epithelium of the alveoli, which regenerates so luxuriantly), and in addition, branched and spindle-shaped cells in great quantity. The whitish-yellow deposits are sometimes like isolated granules, of the size of the finest gravel; sometimes closely compacted granules, of which agglomerations or compact masses are made up of the size of a pea or larger, having an irregular form and indefinite outline. They are composed of a yellow, dry, friable tissue, destitute of blood and air, and, by virtue of their stiffness, do not sink in upon section, but project. According to their size, they correspond either to single alveoli, or more often to whole lobules, or a smaller or larger assem-

blage of lobules—indeed, even to the major part of a whole lobe uninterruptedly; yet they seldom have well-defined borders, but pass over into the red ground, generally losing themselves in points. Microscopically examined, the yellowish deposits resolve into shrunken alveolar epithelium, or their remnants (destroyed nuclei and molecules), as well as into necrotic framework itself; hence we find in the connective-tissue tracts and vessel-membranes more or less fat granules, generally distributed in rows. *Pus corpuscles or their remnants are nowhere to be found, or at most, there are traces of them in the lung-parenchyma*, probably carried over from the bronchi. Every one will see that the red ground corresponds to our fresh genuine desquamative pneumonia, and that the whitish-yellow deposits indicate only anæmic necrosed parts of the same.

Whence, therefore, arises the remarkable difference—whence the absolute capillary anæmia leading to necrosis? I stated, in my last lecture, that the chief point of the process in desquamative pneumonia lay in the swelling of the stroma containing the vessels, of the alveolar walls, and of the interstitial tissue. The changes in the epithelium and its desquamation, although for the investigator at the bedside and the post-mortem room the better marked, are still rather secondary, and depend directly upon the change in the lung framework. If the swelling in certain vascular parts be considerable and increasing, there may come a time when the capillaries will become so much compressed that they will be deprived of the current of blood; and since we have not simply to deal with watery swelling, as in catarrh and croup—where the serum easily exudes from the injured connective-tissue of the stroma—but with a compact, gelatinous, albuminous, plastic infiltration, with development of spindle-shaped and stellate cells, it will rather surprise you that desquamative pneumonia does not *always* lead to anæmic necrosis, that it does not *always* have this course and result. Indeed, the *pure* genuine desquamative pneumonia, the form for which I,

κατ' ἐξοχήν, will reserve this name, is a great rarity, thus contrasting with the frequency of cheesy pneumonia. There must exist then a cause whose rare absence results in pure desquamative pneumonia, and whose more frequent presence occasions cheesy pneumonia, a cause which does not at all change the proposition that, in effect, both conditions are only modifications of one and the same disease. This cause is—*independent of the embryonal connective-tissue hyperplasia which, without this, already characterizes genuine desquamative pneumonia—a development of cells with exuberant small shining nuclei, accompanying the finest arterial twigs externally, seated in their adventitia and causing this to swell up into lumps by reason of its irregular force, sometimes losing itself diffusely, and again rising up diffusely.*

In the whitish-yellow deposits, the cell growth can be demonstrated only in the earliest condition on account of the fat molecules which appear so soon; on the contrary, in the red tissue, especially in the immediate neighborhood of the yellow granules, we can be sure of finding them.

There is no doubt that this cell-development, although at the beginning it may be so obscure, is that which surrounds the capillaries and so brings on anæmic necrosis. Its amount regulates the extension of the necrosis, and I must repeat that it is seldom so small that the disease remains a pure genuine desquamative pneumonia, *i. e.* one which does not become cheesy. It is also the cause of the "adhesive" infiltration, being much thicker and more gelatinous, and gives the reason also for limiting the term "gelatinous pneumonia" to cheesy pneumonia. One more term is to be spoken of, the so-called *white pneumonia of the new-born*" in Pemphigus. This is different from the cheesy pneumonia, for the lung-framework is infiltrated with growing connective-tissue cells which show two or three nuclei. The alveolar epithelium is partly in a state of fatty and myeline degeneration, partly occupied with regenerative growth, so that we find various large cells. But I wished only to give the basis of cheesy pneumonia, arising upon

and out of desquamative pneumonia, and will return later to the significance of the cell-development just described, and to the correctness or incorrectness of the former practice of calling the yellow deposits *tubercle,* and the whole disease *tubercular pneumonia.* I myself formerly described the process leading to anæmic necrosis under the name of diphtheritis, and in so doing, only employed the previously used nomenclature. In my work upon diphtheria * I have, on the other hand, in view of the fact that diphtheria is a peculiar and specific disease, sought to remove confusion by the proposal to strike out the name "diphtheritic inflammation," for sloughing is not an inflammation, and in its place to speak only of slough-production, of anæmic-necrosis and dry gangrene, and to indicate the cause of the sloughing by means of an additional word. Yet you will find that before this (1856) I had a correct conception of the essential part of the pathology of the process. We may have, then, in the acute stage of cheesy pneumonia, a necrosing desquamative pneumonia, which term is not calculated to prejudice, and perfectly characterizes the situation.

No other condition ought to be confounded with the disease in question. If, for example, from a theoretical stand-point it is not to be denied that cheesy degeneration may occur in catarrhal pneumonia and in very rare cases of croupous pneumonia, yet these inflammations do not deserve the name "cheesy pneumonia." The diagnostic reason is that in catarrh and croup the lung-framework itself does not become cheesy but only the alveolar contents, the cell development in the sheath of the arterial capillaries, the increase of spindle-shaped and stellate cells, and the gelatinous infiltration are wanting, and the alveolar contents also consist almost solely of pus-corpuscles and mucous or fibrine, and not of desquamated alveolar epithelium. *Neither from catarrhal nor croupous pneumonia does cheesy pneumonia develop,* but solely and alone from necrosing desquamative pneumonia.

* Zeitschrift für Biologie, III. p. 341.

B. *The subacute and chronic stage.*

This is characterized by retrograde or terminal processes, which, nevertheless, do not appear in equal degree in all parts of the diseased lung. The whitish-yellow anæmic sloughy spots, if they attain to the size of a pea or more, remain unchanged, or only lose their indefinite borders and irregular shapes, and so are transformed into sharply defined centres, then through continual loss of water they shrink more and more and become cheesy. Meanwhile it is the red ground, the part affected with desquamative inflammation, which, through temporary fatty degeneration, may be brought back completely and quickly into its normal relation.

This is the first possible issue, and it occurs the sooner, the less the number of the yellow spots, and the more widely separated they are. The lung, then, contains, in otherwise perfectly sound tissue, cheesy patches, in multiple diffusion, which evidently surround a bronchiole.

If we abide by our conception and mode of origin of lobar and lobular, these multiple cheesy spots would be lobar according to their appearance and size, but lobular according to their mode of origin. They would correspond, indeed, to a cluster of terminal bronchial twigs, because the parenchymatous inflammation may, and in fact does appear more violently and powerfully in peribronchial, than in interalveolar tissue, but they would not have developed from the bronchi, but from the lung-tissue. In a given case it would be indeed impossible, in the return of the lung-tissue lying between the spots to perfectly normal condition, to distinguish the centres rescued from cheesy pneumonia from the genuine lobular centres.

Although Waldenburg in his noble work* observes, "it is now shown by evidence that the cheesy spots have never passed through a stage of grey infiltration, and it is yet more certain that they arise from pus, originally thickly fluid and later inspissated," yet in spite of his positive assur-

* Die Tuberkulose, etc., p. 64.

ance I must contradict this. For the cheesy centres in question proceed in the present case from tissue-sloughs, which never contained pus, and the grey jelly-like infiltration, which I have already described, belongs in fact to desquamative pneumonia, the forerunner of sloughing.

I will, in my next lecture, speak of the genuine lobular cheesy centres, arising from the bronchi, which proceed from purulent infiltration, and apparently cause confusion. These have nothing to do with cheesy pneumonia, and just as little also with catarrhal pneumonia.

If the desquamative pneumonia between the cheesy spots does not pass to complete resolution (and this most frequently is the case), there are still two results possible —either the peculiar issue of desquamative pneumonia resulting in thickening and pigmentation (cirrhosis), as has been explained, or chronic fatty degeneration, which latter is distinguished from the former by pallor, lack of blood, less thickening, and feeble pigmentation of the lung framework.

We can sometimes recognize a combination of all three results of the desquamative pneumonia between the multiple cheesy spots—restoration, fatty degeneration, and thickening. No difficulty exists any longer in considering their origin to be diffuse and lobar. They are only the signs of the unequal division of the cellular infiltration which, in one place, leads to anæmic necrosis, in another to cirrhotic thickening, and in a third to fatty degeneration. A section of the lung shows us again the surprising sight already described in cirrhosis, of granite-like variegation. Close around the whitish-yellow cheesy spots we see the black cirrhosis, in this peribronchitic milk-white fibres or points, and intermingled everywhere the greyish yellow of fatty degeneration. If we add to this the peculiar grey transparency of amyloid degeneration, and here and there the hyperæmic redness which is produced through the collateral supply of blood (the regions in the cheesy spots being removed from

circulation), we have indeed a rich mixture of colors.* That the cheesy spots and the milk-white points have been called tubercle is known, but you will scarcely, after my explanation, apply that name to them. Whether, really, miliary tubercle occurs among them, we will discuss in a later lecture.

In the acute stage of cheesy pneumonia, a fourth result may take place, an important and very influential occurrence, viz. that, in very marked intensity of the process and in significant extension of the sloughing in a continuous row of spots (the spots being seldom smaller in size than a hazel-nut), the whole dead mass, the whole centre may be transformed into a sloughy plug, become loosened through limiting suppuration, and fall away, so that a loss of substance in the lung-parenchyma corresponding to the diameter of the necrosed part is established. As you know, we call the tissue deficiencies thus produced *cavities, excavations, hollow ulcers*, and in especial *lung*-cavities to distinguish them from the bronchial cavities already described in cirrhosis, and others yet to be described.

The necrosis and consequent acute formation of cavities dependent upon the development of cells which ligate the arterial capillaries from without, and cut off the current of blood, presents no isolated appearance in the pathology of lung-diseases. Cavities occur likewise in an acute manner in the lungs in other ways, especially in consequence of the plugging of fine arterial branches. The wedge-shaped pyæmic infarction, formed by embolism, at the outset

* It often happens that a person who bears some trace or other of desquamative pneumonia, falls sick, not with this, but with catarrhal, or oftener with croupous pneumonia, which seizes upon the more or less intact part of the lung, especially the lower lobes. He may get well or die, but in the latter case there can be no difficulty in distinguishing well-marked processes in the affected lung from old changes. We should commit a gross error, if we should interpret such a coincidence as signifying that the croup was a relapse of a former process, and consequently that the still-existing cheesy pneumonia was a sequel of a still earlier croup.

having a hemorrhagic appearance, but soon transformed to a yellow dry slough, may likewise, through limiting suppuration, be loosened as a plug, and come to lie in a cavity or be dissolved in the pus. The lung infarction of Laennec, which originates in thrombosis of dilated capillaries, and, together with brown induration in certain heart affections, appears in connection with wonderful slowing of the blood stream, appears also (as I remarked in the proper place) quite like the gangrene developed from red hepatization in croupous pneumonia, and it may break down into granules when lying in a cavity. In speaking of purulent infiltration proceeding from croupous pneumonia, I mentioned abscess-cavities, and in pure genuine desquamative pneumonia diffuse softening, and I must finally remind you of the formation of cavities whose original cause is situated, not in compressed or plugged blood-vessels, but in the lumen of the bronchi; and I allude here especially to the destructive working of the numerous foreign bodies which, by aspiration, are brought into the air passages.

It is not difficult to discover the processes which were the cause of a given acute formation of a cavity. The examination of the lung itself and the accompanying relations in the body make it easy to recognize the beginning, and you may well permit me to group together their differences.

There always belongs to cavity-formation, and likewise to the preceding necrosis, a stoppage of circulation, and it is just this which the catarrhal and croupous pneumonia do not have. These latter diseases lead to necrosis and cavity-formation, not through their peculiar process, like the desquamative pneumonia, but only through chances and combinations.

To return, however, to cavity-formation, we find that contiguous cavities may break into each other and establish sacculated ulcers; those superficially situated may perforate the pleura and give rise to pneumothorax. The cases in which, in the acute stage of cheesy (necrosing desquamative)

pneumonia, numberless cavities of different sizes have formed, from the apex to the base of the lung, within four or six weeks after the beginning of the disease, are very worthy of note, but they are relatively seldom. We recognize them by the fact that the adjoining tissue still bears in itself the signs of fresh desquamative pneumonia, while in those which have existed longer or originated later, the neighboring tissue is either normal or may show the signs of hidden desquamative inflammation, of chronic fatty degeneration or cirrhosis. We may often observe all the stages of limitation and of gradual separation of the sloughy plug, until finally (just as in pyæmic centres) it hangs at the delicate end of a twig and of its vessels as if on a stem, and floats in the cavity. With increasing suppuration, which of course is only in a measure connected with the extent of the previous loosening of the plug, the washed slough softens at its surface, and finally breaks down into a friable, putrescent, granular mass, which fills the cavity. The cavity is prolonged according to its diameter into a smaller or larger bronchial branch. The whole domain up to the cartilaginous bronchi may belong to such a cavity, and its contents may close and obstruct periodically free communication with the bronchi. We often see such cavities belonging to single bronchial tracts arranged in rows, by which arrangement the cavities form a pear or bullet-shaped figure, the base being outwards, the apex towards the root of the lung. The limiting suppuration and the gradually developing granulations, with a continual envelope of pus (the pyogenic membrane) for the purpose of casting off the sloughy plug or cheesy mass, afford the only opportunity of seeing pus-corpuscles in the parenchyma of lung, affected by desquamative or cheesy inflammation.

As follows from the preceding, the inner wall of a cavity has varying relations: immediately after the acute process by which the sloughy plug is cast off, it is fatty and ragged; afterwards it becomes smooth, coincidently with complete loosening of the sloughy shreds and with forma-

tion of granulations. Still later, the granulations become a fibrous lining layer which characterizes the chronic condition. The granulations may also, by-and-bye, produce pus which breaks down under the influence of the air admitted, and gives a new source for sloughing. Layer after layer of cirrhotic wall then becomes loosened, and the cavity becomes thereby enlarged, blood-vessels previously dilated here and there (aneurisms of pulmonary branches of varying diameter) are opened and bleed. So it happens that cavities above the size of a pea never heal. Only those beneath that size may, by compact plugging and under the influence of progressive cirrhosis, be closed and cicatrize, of course with marked corrugated retraction of the lung-surface.

Cavities originate, however, in cheesy pneumonia not only in an acute fashion. The smaller cheesy centres, provided that their circumference does not exceed that of a pea, shrink more and more by absorption, their mass becomes drier, mortar-like, disappears completely or becomes chalky—even stony—and lies finally in the cirrhotic tissue as in a cicatricial capsule, like the encapsulated contents of a bronchus in which the constituents of the former lung-tissue (the elastic fibres), are wanting. Near the lung-surface they give rise to radiated cicatricial contractions. Larger cheesy centres, on the other hand, instead of experiencing a cicatricial contraction, are rather held fast in their periphery by the cirrhotic traction-force acting outwardly, their substance becomes moister or even softened, and then the fibrous capsule surrounds a chronic lung-cavity; and this, being the same as the acutely formed cavity which has become chronic, enlarges continuously in the same way. It is very like those cavities which develop from hypertrophic bronchiectasis. The chronic are distinguished from the acute by being furnished with a cirrhotic capsule from the outset. Neighboring chronic cavities may break into each other through gradual enlargement, and lead to sacculated ulcers, to perforations in the larger

bronchi, and in the pleura with pneumothorax. Pneumothorax is, nevertheless, in acute cavity-formation considerably more frequent than in chronic cavity-formation in which the cirrhotic surroundings oppose a certain amount of resistance to rupture.

A small number of cavities, and especially a single one, is less often found than a large number. In acute formation the number is usually great and the diameter small; in chronic formation the number less and the diameter greater, even to the size of the fist or larger. Apparently the impression upon the vital powers is small in the chronic formation of a cavity.

In the cases having only a few cavities or only one, it is remarkable that these occur by far the most often in the apex of the upper lobe, or in its immediate vicinity, and most seldom of all in the lower lobe. Hence comes the unanswerable argument that not only is the intensity of the inflammation in the upper part greatest, diminishing as we go downwards, but that it in fact begins above and spreads downwards; indeed, in many cases, it is confined to the apices.

This fact is to be used in tracing back with certainty to their origin, chronic cavities whose ætiology seems obscure or hidden; for cavities from lung-abscess and moist gangrene —from infarction and foreign bodies—are located rather in the lower lobes, and lack those traces of former chronic processes which we find in the upper lobes; while, on the other hand, cavities from cheesy pneumonia, when they occur in the lower lobes, scarcely ever exist without disease in the upper parts, *i. e.* without cavities, cheesy centres, fatty degeneration, thickening, pigmentation, etc. It must not be forgotten that the contents of cavities may be transferred by respiration from one bronchial branch to another, from one lung to another, and may occasion lobular necrosis at the spot where they are arrested and impacted.

LECTURE VIII.

Peribronchitis.

Although my lectures are properly devoted to inflammation of the lungs, still it is impossible to fully understand parenchymatous pneumonia, without at least a short description of the oft-mentioned *peribronchitis*, since this forms not only many combinations with the different grades and stages of desquamative pneumonia, but is also the cause of an equally important disease, *lobular pneumonia.*

By peribronchitis we do not understand an inflammation of the bronchial tube, which, like the catarrhal and croupous forms, shows its most prominent characteristics on the surface, in a qualitative change and quantitative increase in the secretory activity of the mucous membrane; but we mean an inflammation which, attacking the tissues of the bronchial walls, has its seat particularly in the adventitia of the small and very fine tubes, rising also to the larger and cartilaginous branches.

We find its analogy in a parenchymatous inflammation of the lungs, where there is an invasion of the walls of the alveoli, their interstices, and the interlobular connective tissue. From its situation it generally gets the additional name of "*capillary*" peribronchitis or peribronchiolitis.

From the character of the inflammation it is possible that the mucous membrane may be intact. It may, however, be simultaneously attacked by either a catarrhal, croupous, or ulcerative inflammation; and if there is nothing to lead us to a contrary conclusion, this may generally be considered to have occured. As in parenchymatous pneu-

monia, so also here we must distinguish two forms, dependent on the occurrence or absence of suppuration; they are peribronchitis simplex and peribronchitis purulenta. Both forms may exist together.

A. *Peribronchitis simplex.*

This form follows the chronic stages of desquamative pneumonia, particularly cirrhosis, and *is always a chronic inflammation.* We can distinguish several subordinate forms.

1. I have already mentioned, for example, *peribronchitis fibrosa* (Lecture VI.), which accompanies cirrhosis, and which by an hypertrophy of the connective tissue, follows the bronchioles in white fibrous bands, even in some cases to the extent of causing their obliteration.

We would in this case include the whole under the title of diffuse cirrhosis of the lungs; that is, we would include the fibrous peribronchitis, provided the process of obliteration of the alveoli and bronchioles and the filling of the space by fibrous tissue is pretty widely extended. We speak also, as I have already explained to you, of a *lobular cirrhosis*, when we find in the midst of healthy lung-tissue multiple black fibrous nodes, varying in diameter from one to one and a half inches.

Finally, we use the name *peribronchitis fibrosa* where a portion of the lung is invaded by tree-shaped branched fibrous bands of interstitial connective tissue. Whether we must acknowledge the multiple fibrous nodes which lie between diffuse cirrhosis and true fibrous peribronchitis to be lobular or lobar in their origin, is often concretely very difficult to decide. In general it may be said that the smaller the nodules, the greater the probability of their being lobular, although the size is not a proper criterion for a definite decision. The question is, however, easily answered if we remember that the peribronchial, interlobular, and alveolar connective tissues are continuous and histologically similar, and that there are no such differences between

them as are found to exist between the inner surfaces of the bronchi and alveoli. In fact, diffuse cirrhosis also produces multiple black fibrous nodules, while true peribronchitis fibrosa forms simply bands, which are exactly similar in composition but dissimilar in quantity, and are only distinguished by the fact that in one the interalveolar and in the other the peribronchial connective tissues are more hypertrophied. All three forms, distinguished by these differences in position and extension, may occur side by side in one and the same lung.

2. Another form of peribronchitis is that which has lately been called *peribronchitis nodosa*, and which is one of the *most frequent diseases* met with in the respiratory organs.

A higher grade of desquamative pneumonia, as you have already seen, shows besides, a development of connective tissue elements, a cellular proliferation of the endothelium of the lymphatics belonging to the arterial coats.

Peribronchitis nodosa proves to have a similar origin, for in the external layers of the bronchial walls is developed the same cell-growth with the same irregularities in thickness. The bronchus becomes thus irregular, knotty, and thickened, the thickened tissue being fibrous and containing endothelial cells.

A bronchus affected in this manner, if cut either square or diagonally, shows the thickening, as a rule, to be not regular on its whole circumference, but generally greater on one side than on the other. If the tube is cut longitudinally the cut surface of the thickened portion is spindle-shaped.

Only the finest bronchioles are obliterated; in those of a large calibre the lumen is recognizable and generally quite empty and free from secretions, showing as a hole in the section of the nodule; or it may, on the contrary, be filled with the products of catarrhal or croupous processes, which may be either soft, or dry, cheesy, and easily pressed out and detached.

If the thickening is confined to the finer bronchial branches, the nodules will appear, on a freshly cut surface

of the lung, discrete and often very widely separated. If, however, the very finest bronchioles are the parts attacked, and even the infundibula is invaded, we shall see, according as the cut is at right angles or perpendicular, either cross-shaped or tree-shaped groups and collections of nodules.

We can readily understand that both the discrete and the grouped (gregalen) nodules may be observed in the same lung.

The color, if the thickening has changed into fibrous tissue and its cells have undergone fatty degeneration, is grey, opalescent or yellowish.

Since this process is chronic, we formerly gave it the name of "*chronic discrete* or *aggregated miliary tuberculosis*," instead of nodular peribronchitis. For just here a very gross error slipped in: the purulent cheesy contents of a nodular bronchial tube were mistaken for the central softening of a tubercle, and important conclusions drawn as to the properties and course of tubercles.

To-day no one will make this mistake, for we now seek for the chronic tubercles in the nodular thickening of the bronchial walls, and we know that the chance contents of a bronchus has nothing to do with the substance of the newly formed node.

The microscopical appearances of nodular peribronchitis and chronic miliary tuberculosis are very similar and can be very easily confounded; you can, however, only confound them histologically and histogenetically, and not so easily from a physiological and pathological stand-point. I will touch again on this point later.

Peribronchitis nodosa sometimes occurs as a simple uncomplicated disease. In this case the nodules are solid, of a greyish-yellow color, and are found in the midst of perfectly healthy tissue. It is, however, generally accompanied by P. fibrosa, that is, the nodules spread into fibrous thickenings with black streaks.

The multiple black fibrous nodes are beset almost regularly in their whole circumference by nodules, which

appear on section like a grey circle of pearls surrounding the black node. We are forced by these appearances to the conclusion that peribronchitis nodosa reaches the alveolar parenchyma, and, by an hypertrophy of the interalveolar tissues, goes over into it; that is, the connective tissue participates by being changed into multiple black fibrous nodes. It is seen also as an extension of a diffuse cirrhosis of the lungs, both in the aggregated and discrete forms.

Both the fibrous and nodular peribronchitis, insomuch as they tend to cause an obliteration of the finer bronchial tubes, can by an increased shrinking of the thickened tissues, cause an *atrophic ectasis* of the *alveoles* and *bronchioles* in the adjoining lung tissue, and also of the surrounding capillaries. In very severe and acute cases, an absolute stoppage of the local circulation commonly follows; the increased cell-growth, by its increasing pressure, diminishing more and more the circumference both of the smaller branches of the bronchial artery and the capillaries themselves. Thus we may have as a result of these changes, not only a fatty degeneration of the nodule, but also an anæmic necrosis, and possibly even a casting off of a slough-like strip from a bronchial wall (P. nodosa *necrotica*). More commonly, however, the necrosed portion remains relatively small, and being surrounded by organized envelopes, simply undergoes a cheesy degeneration (P. nodosa *caseosa*). This is the form which occurs more frequently in the infundibula and bronchioles, and which, by extending the same processes to the alveolar parenchyma, produces another form (not the commoner) which we call *lobular necrotic cheesy pneumonia* (the *chronic disseminated pneumonia* of Lebert). Even although the sloughing process be acute or subacute, still the cheesy degeneration is somewhat chronic in its course, and the lobular centre may, as in diffuse cheesy pneumonia, by-and-by be surrounded by a black cirrhotic tissue, and, if it is quite small, may finally be entirely absorbed.

Multiple lobular pneumonia has been classified under

the title of chronic tuberculosis, and described as a particular, late stage, viz. the stage of cheesy metamorphosis of the tubercles. You will see from later descriptions that the comparison fails in every point. It much more nearly resembles a transitional intermediate form, between diffuse cheesy pneumonia and nodular peribronchitis. For it is, as I have said, the same connective tissue which is attacked in each case, only sometimes the interalveolar and sometimes the peribronchial is the more involved. The causes which lead to the special locations of the process are found in the relative length of its duration, in each case. The most acute forms are generally alveolar, the sub-acute attacks the bronchioles, and the chronic, whether as the remnant of an acute attack or as such from the beginning, implicates the tubes which are not quite so fine.

In this we have a very important guide for our *prognosis;* since manifestly the chronic forms, that is, nodular and fibrous peribronchitis, have much less significance than inflammation of the lung proper; for the P. nodosa, whether aggregated or discrete, is, if considered by itself alone as a local disease, a perfectly harmless affection.

3. There is one peculiar form which I must yet add, to the list; that is, the *lardaceous peribronchitis.* It presents lobular spots or points in the lungs, since it attacks the finest bronchi and from them spreads to the alveolar walls. The appearances which I have so far observed in lardaceous pneumonia are peculiar. The pneumonia follows the smaller arteries in tree-like branches. The section is grey, transparent, firm, nearly free from blood, glistening, and has a lardaceous jelly-like appearance. On microscopic examination we find growths, consisting of small cells in the midst of parenchyma, whose epithelium is in a state of fatty degeneration. The lardaceous points or nets combine with fibrous, nodular, and cheesy lobular pneumonia and peribronchitis.

If desquamative pneumonia is the localized expression of a general disease, and if peribronchitis is, on anatomical

grounds, identical with it, merely having its seat in the peribronchial instead of the interalveolar tissues, then is *peribronchitis an expression of the same general disease*, has the same pathological significance, and gives rise only to a local and moderate danger. There is, however, another form, which is, comparatively speaking, as dangerous as any disease with which we are acquainted, and that is :

B. *Peribronchitis purulenta.*

Notwithstanding its frequency, this disease is very little known, and is certainly not estimated according to its importance. Rindfleisch * has been the most prominent in calling our attention to it. I cite him in order to have authority and support in separating it from the other varieties of peribronchitis ; in describing it as a distinct form of disease, and in not considering it merely as the concomitant of other processes. It occurs as an *independent, uncomplicated* disease.

Its most important characteristic is a *purulent infiltration* of the finer bronchi, especially those of the size in which the cartilage, muscles, and glands are gradually disappearing. The infiltration invades the whole thickness of the bronchial wall from the external coats to the mucous membrane, and extending thence into the interlobular tissues, even forces its way into the alveolar walls.

On section, the bronchus presents a yellowish color, is friable, and the mucous membrane is almost of a fluid consistency, here and there so completely softened that the inner surface of the wall presents ulcerations, losses of substance of greater or less extent ; in some cases strips of mucous membrane, and even of the whole bronchial wall, may be cast off (falsely taken for croupous or diphtheritic membranes), or there may be present a putrid purulent fluid.

The neighboring alveolar parenchyma necessarily takes part in the process, and the bronchi are surrounded by abcesses and ulcers.

* Pages 334 and 345.

Vegetable organisms of the lower orders are always to be found in the ulcerated spots and even in the collected infiltration.

From these circumstances we might apply to this disease the name of *exulcerative* peribronchitis.

The purulent softening ought not and must not be confounded with anæmic necrosis, although the purulent infiltration, and the portions of purulenty softened tissue, in which only the elastic fibres are still to be recognized, can in addition become inspissated and as dead masses undergo cheesy degeneration; or, if the circumference is great enough, may lead to the formation of cavities similar to those found in anæmic necrosis.

Purulent peribronchitis shows itself in the lung parenchyma by *yellow lobular purulent and cheesy masses, lobular infiltration and necrosis* (the commonest form of chronic disseminated pneumonia), as well as by *cavities resulting from ulceration.*

When a cavity is formed, either the terminal branches of the bronchi, together with the adjacent lobuli, are dissolved in the pus, or the purulent infiltration, and with it maceration of the bronchioles, extends still further, and only cease when it has reached the larger bronchi, which are provided with cartilaginous rings, and which then come sharply to an end, being as it were gnawed off. Generally then ulcerative changes are preceded by a bronchiectasis which is formed as a result of the following process. The purulent infiltration destroys the bronchial muscles and pulls back eccentrically the neighboring elastic alveoli which are attached to the bronchus. In this case, just as in hypertrophic bronchiectasis, the empty space is a bronchus, and existed before the ulcerative process began; the difference being simply that in hypertrophic bronchiectasis the walls are from the beginning thickened by a cirrhotic process, and here are yellow and purulent, while on the other hand, in the lobular necrosis and in lobular ulceration both the cavity, and the loss of substance by ulceration, begin

together; the cavity first appearing through the loss of substance.

It stands to reason that, as in desquamative pneumonia under the influence of the parenchymatous swelling, the bronchial and alveolar epithelium is destroyed by fatty degeneration and desquamation, so here, under the influence of the purulent infiltration of the bronchial mucous membrane and, particularly in the finer bronchi, of the whole bronchial wall, also of the walls of the infundibulæ and alveoli, the same process must take place, though in contradistinction to desquamative pneumonia the epithelium here perishes without any evidence of proliferation or attempts at regeneration. For in genuine desquamative pneumonia, the parenchymatous swelling has a productive character, but here it is only destructive in its tendencies.

We see, then, that the purulent infiltrated and softened portions of tissue include also degenerated epithelium.

At this point I must call your attention to an important clinical feature, which is that the infiltrated mucous membrane ceases to secrete and is more or less dry, and that only when the ulcerative process reaches the surface, can a putrescent purulent fluid flow into the bronchial tubes and be coughed up.

You can now easily see the differences between this and other inflammatory processes. Catarrhal pneumonia, which like the purulent peribronchitis occasions the formation of lobular centres in the lung, being only a superficial process, lacks the purulent parenchymatous infiltration, the complete desquamation of the epithelium, dryness of the surface of the mucous membrane, and purulent liquefaction and cheesy degeneration of the parenchyma. In croupous pneumonia the purulent infiltration is lobar, and we can make no mistake as to the character of the process even at the first glance.

But how are we to distinguish it from cheesy pneumonia, particularly in its chronic stage, since desquamative pneumonia is always lobar, and cheesy pneumonia has a yellow

color and an uneven distribution of necrosed portions throughout the affected part? The answer is not difficult, since cheesy pneumonia, like genuine desquamative pneumonia, has no connection with purulent infiltration; and purulent peribronchitis, considered according to its duration is properly a *subacute inflammation;* moreover, the cheesy centres which may appear in its course are easily referred to their proper origin by their not having any cirrhotic capsule. For in purulent peribronchitis the surroundings of the affected lobule may be quite normal, while in cheesy pneumonia, in the acute stage, there is always some desquamative inflammation. I am quite certain that hitherto, purulent peribronchitis has been generally confounded with, and mistaken for cheesy pneumonia, particularly if the object in view was to prove the development of the latter from bronchitis.

Finally, purulent peribronchitis is to be distinguished from pyæmic interlobular pneumonia by the latter always presupposing an infection, by its being lobar, and from the fact that it does not confine itself to the bronchial walls, but, on the contrary, from the very start follows the lymph-vessels between lobules, and extends under the pleura, dividing it in a certain degree into districts by a network of yellow lines.

There are still other very interesting appearances which are observed in purulent peribronchitis. In consequence of the swelling and thickening of the bronchial tissues, the entrance to the air-filled alveoli are often suddenly stopped up. The result is a *multiple lobular emphysema*, for, as a rule, although no more air can find its way out on expiration, yet on inspiration a little can still be admitted. This emphysema is almost always found as a complication of purulent peribronchitis if the latter has extended into the lung-tissue proper. A whole lobule may thus become distended to the size of a bean or walnut, its vertex extending on the outside nearly through the pleura. The surrounding tissues also are flattened, while within they are smoothed

out through a destruction of the alveolar walls, which would otherwise protrude. The purulent yellow softening infiltration extends from the adjoining lung parenchyma in a circle around the sides of the air vesicle, which is thus made to protrude above the normal contour of the surface of the lung,* thus rupture and entrance of air into the pleural cavity (pneumothorax) are made imminent; and since the process attains the same degree simultaneously in many points, and there is the same danger of rupture in each, there may be many ruptures in existence at the same time, each aiding in the production of the pneumothorax.

Excepting traumatic causes, and all chronic processes, since we are now dealing only with the acute or subacute, I know of no more frequent cause for pneumothorax than purulent peribronchitis. Neither necrosing desquamative pneumonia, pyæmic embolism, gangrene of the lung, nor foreign-body pneumonia are so frequently met with in this relation.

Since purulent peribronchitis involves the interlobular connective tissue, it may extend also into the subplural connective tissue, and may, in this way, just as well as by the emptying of the fluid contents of the flattened emphysematous vesicles, lead to a *putrid purulent* form of pleurisy, *pyopneumothorax.* The collapse of the emptied air-vesicle, the layer of exudation, and the compression of the whole lung, very often so completely cover over the point of rupture, that on post-mortem examination we are unable to find it, especially since, in general, other suspected emphysematous vesicles with purulent walls are close to it. The layer of exudation not only answers the purpose of shutting up and gluing together the ruptured vesicles, but it also prevents the rupture of those near by.

The occurrence of lobular emphysema as a result of purulent infiltration, and still more the intervening unaffected portions of alveolar parenchyma leave no doubt that the

* Large bladder-like protuberances of the lung pleura. Rokitansky, III. 93.

process spreads from the bronchial tubes into the healthy air-containing portions of lung tissue. We must notice also the collateral processes, such as vacillation in the blood supply, there being sometimes anæmia, again congestion, and even hemorrhage.

The clinical diagnosis, so long as pneumothorax and pleuritis do not complicate the case, can be indicated and decided by a clear, low-pitched, more rarely dull percussion note, catarrhal rales, with long-continued fever, and purulent, even fœtid sputa.

It is a very rare occurrence for a purulent peribronchitis to become chronic, or even to diminish in activity. If it does last a long time it takes on a course similar to that of desquamative pneumonia; that is, it spreads below the boundaries of the previous affection by repeated relapses and subsequent extensions, with *cheesy thickening* of the *purulent infiltration* and slight thickening of the layers of connective tissue.

Only, the extension from above downward, even if it is the commoner, is not certainly discontinued. It proves fatal when the extension has reached a certain degree, or sooner through the advent of pneumothorax.

It was purulent peribronchitis which I mentioned, when speaking of cheesy pneumonia in a previous lecture, as being almost the only case in which cheesy centres (lobular necrosis) follow purulent infiltrations; and it appears to have been used as a basis in the investigations of cheesy pneumonia, since we invariably assumed that this arose only from pus. This disease also makes combinations with desquamative pneumonia and nodular peribronchitis, and often causes the closing scene in the sad drama of life. The well-known sentence which Niemeyer made use of in respect to the combination of tuberculosis and phthsis I will here somewhat modify: "*The greatest danger in chronic inflammatory processes in the lungs is, that purulent peribronchitis may be developed.*"

It is also a fact worthy of notice how at one time pur-

ulent peribronchitis occurs alone and independent, and how at others it has a tendency to combine with other inflammatory processes where there is no formation of pus. If you bear in mind that the plastic activity in desquamative pneumonia and nodular peribronchitis, consists in an exuberant proliferation of connective-tissue cells and endothelium of the lymph canals, that this growth is by a division of the cell nuclei, which again become fixed tissue elements, you will not be able to admit the explanation of the formation of pus simply in an emigration and infiltration of white blood corpuscles—(we must also remark here that the wandering cells do not reach the uninvolved surface of the mucous membrane)—but you will, on the contrary, I think, be driven to the conclusion that the pus corpuscles must be formed in and on the spot. For the connective tissue cells and the endothelium of the lymph canals do produce these pus cells free, within themselves (endogen) from their nuclei, after which they quickly perish and set them (pus cells) free

With me the idea is continually gaining ground that the formation of lymph cells and white blood corpuscles takes place normally from the endothelium of the lymph canals, being, however, confined to certain localities in the organism, that is, to the so-called lymph and blood glands; under pathological irritation however, that they may be produced wherever there are lymph canals, or connective tissue corpuscles analagous to endothelium, or even from the venous capillaries alone. The change from the formation of fixed tissue elements to the development of free (lymph pus) cells is always a bad sign, for the result is a destruction, instead of an hypertrophy of tissue.

LECTURE IX.

The Tubercle.

In my previous lectures I have endeavored not only to explain to you the pathological anatomy of the different forms of inflammation of the lungs, but also to introduce you into the varied and interesting field of their pathology. By so doing I have also accomplished the subordinate purpose of laying a foundation for a clear representation of tuberculosis and consumption of the lungs, and you will find that I have thus made it much easier for myself, and have quite smoothed over the ground, otherwise so difficult, which is covered by these diseases.

If I ask you to examine with me the *tubercle* somewhat more exactly, I must connect the examination with what has already been said of inflammatory processes, by proposing now, as the proper time, the question, pathologically not unimportant, whether the formation of tubercles really depends on inflammatory processes or not. And this question will be discussed with great earnestness.

Although you are doubtless sufficiently informed as to what one understands by a tubercle, still I cannot avoid stating in a few compressed sentences what our present knowledge and views really are.

A *tubercle* (miliary tubercle, Bayle; granulation, Louis, Empis) is a new growth, varying in size from that of a microscopic object to a diameter of $\frac{1}{12}$—$\frac{1}{6}'''$; in itself without vessels, but surrounded by a vascular network. It has its origin in the connective tissue which contains the juice canals and lymphatics, and particularly in the adventitia

of the finer arteries and its lymph-sheaths, and from these points it emerges as a well-organized structure. It shows in its finer organization an analogy with the lymphoid organs of the body (*tubercle lymphoma*), such as the malphigian corpuscles of the spleen and the lenticular glands of the intestines. E. Wagner has proved by his most careful investigations* that this analogy, which was urged by Virchow and myself more than sixteen years ago, amounts nearly to identity.

On microscopic examination we recognize, as a rule, a rounded body; and in general, instead of one, we recognize two or three or even eight or ten such bodies to a certain degree shoved into and upon each other, so that properly the supposed tubercle appears rather to be composed of a group of spherical bodies, or as if there were many spherical segments pressed together.

As to its histology, E. Wagner has determined the existence of a fine meshed network (reticulum, *reticulated tubercle*), in which there are distributed cells which increase both in number and size toward the centre. In quite young, recent tubercles we can recognize, without doubt, two sorts of fibres.

First, *connective tissue bands*, which run on the periphery of the sphere, are thick, parallel, cross-shaped, at least representing the segment of a cross, and in which we can recognize the small, round, glistening nuclei of a cellular structure, and here and there a few vessels.

Second, the *reticulum proper*, which extends toward the centre of the body, and which I am obliged to consider, from its want of nuclei, as a homogeneous connecting substance, and not as a true connective tissue; for though in its nodular points there are fibre-branches rising above the general level of the field, which give the illusive appearance of nuclei, I have never been able to see in it anything which I could declare to be true nuclei. This reticulum is entirely without vessels. It is also particularly to be

* Das tuberkelahnliche Lymphadenom, 1871.

noted that the younger the tubercle the softer the substance of the reticulum, and in its very first stage the network is entirely wanting, the cells lying directly one on the other.

The cells of which it is composed are not all of the same kind. I distinguish, *first*, the *cytoid bodies*, these being chiefly found in the layer of connective tissue. The opinion that these elements of the tubercle are lymph or colorless blood corpuscles is held by very many authors. But a comparison made in the same organism, between the corpuscles of the tubercle and those taken directly from the blood or lymph, shows unmistakable differences; for the former cells, and particularly their nuclei, are much smaller and more glistening than those from the blood or lymph. We cannot account for this difference in size by the difference in the menstruum, since those blood and lymph corpuscles, which have wandered through the walls of the vessels into the parenchyma of an organ, retain not only their own size, but particularly that of their nuclei, unchanged. Neither can we consider them as the young precursors of lymph corpuscles which have not yet reached their full size, since tubercle cells never, at least as far as relates to their nuclei, become larger; following, on the contrary, an opposite course in their development. Finally the whole form, organization, and further changes in the tubercle contradict the theory of a simple congregation of normal lymph corpuscles. I must, however, return to this question again later.

Secondly, there are the so-called *giant cells* (Langham's). They have a finely granular,* grey or brownish protoplasm, branched-like processes, and numerous nuclei congregated in their centres. There is no doubt that the abundance of the nuclei which they contain is produced by division. These nuclei are round or oval, nearly all of the same size, but always larger than those of a lymph corpuscle, and consequently larger than the nuclei of the above-mentioned cytoid cells. The giant cells lie near the centre

* With reference to these granules, see page 121.

of the reticulum; there is often only a single one, or if there are many, the one nearest the centre exceeds in size those which lie around it. It is thus distinguished as the *mother cell of the tubercle;* and recent observers have, from this circumstance, with a certain authority, laid great stress on its existence as a characteristic of tubercle. Schüppel,* however, goes too far when he declares it to be constant and—only without using the expression—specific; for there are tubercle-lymphoma in which we seek in vain for the giant cells, at any stage of their existence; and in others, if a giant cell develops early, it perishes later on, and in this way we may fail to find it. Again, it is difficult to discover genuine histological marks of distinction—that is, belonging to the cell itself, between other well-known giant cells (for example, those on the inner surface of the periosteum, in growing bone, in myeloid sarcoma, true myoma, epithelial new growths, etc.) and those which we find in tubercle-lymphoma, even supposing them to be isolated or examination.†

Finally, besides the cytoid and giant cells, there are cells of an *epithelial character*, which, judged both by their size and position, are in a transition state between the other two varieties. Their protoplasm is glistening, finely granular, and enveloped in a membrane. They have either one large or many small nuclei, which sometimes resemble those of the *giant cells* and sometimes those of the cytoid bodies. It seems to me to be beyond doubt that they have the same nature as, and a similar origin with the giant cells; but that they begin to develop later, and so cannot reach the size attained by the giant cells. The smaller giant cells are the largest cells of this sort. I have mentioned already that they fill the meshes of the reticulum around the giant cells; the nearer they lie to the connective tissue layer the smaller they are; that is, the reticulum becomes closer, and they appear to be lost among the cytoid cells on one side, just as they are lost

* Tuberculosis of the lymph glands, 1871.

† See, however, page 127.

among the giant cells on the other. Where these latter cells have never existed, the centre may be formed of cells of an epithelial character; or these may be wanting, and in that case the tubercle-lymphoma cannot be distinguished from normal lymph follicles.*

As long as the tubercle continues to grow the giant cells continue to form new nuclei, and new cells are produced around them. The reticulum also must increase, and the connective-tissue envelope must become thicker by an increase in the small cells contained in it.

This growth is, however, very circumscribed and short-lived, for the supply of nutrient material is soon exhausted, and then the giant cells partly perish by *fatty degeneration* and partly become hardened and *horny*, and often form laminated concretions (Schüppel). The other cells contained in the reticulum also undergo fatty degeneration. Not so, however, the cytoid cells; they produce an *hypertrophy* of the *connective-tissue envelope* which gradually contracts tighter and tighter around the degenerated central cells.

The formation of a tubercle-lymphoma is accompanied by an *increased formative activity in all the surrounding tissues.* If we regard only the properly reticulated portion as the lymphoma, and consider the connective-tissue envelope as its immediate surrounding, then the participation of this connective tissue in the special activity is shown by its containing the cytoid bodies and by the formation of new vessels in its periphery. Such connective tissue is commonly called "cytogenous." From just this circumstance it is plain that the cytoid bodies are not wandering cells, but that they must have had their origin in a growth of the connective-tissue cells, since they are finally

* Schüppel has lately come to the conclusion (Virch. arch. Bd. 56, p. 44) that the tubercle must be stricken out of the category of lymphoma. The above-given differences (repeated on p. 95) do not disturb the established analogy between tubercle and lymph follicles either anatomically or still less in its pathological relations.

employed in the formation of new connective tissues. The external (*epithelial*) and internal cells of the surrounding *parenchyma of the organ* are also included in this increased formative activity; for they enlarge, both their protoplasm being augmented and their nuclei increased. If the tubercle ceases from further development, the activity in the surrounding elements of the organ also ceases, and they undergo fatty degeneration and absorption; so that under favorable circumstances the tubercle lasts much longer than the surrounding growth which has originated with it. It cannot be argued from this that the tubercle excites and supports the growth, but rather that on its origin all the tissues in which it originates are in certain directions actively implicated.

Tubercles owe their existence to a peculiar method of formation. Although they belong to the lymphatic system and their structure is analogous to that of the lymphoid organs, still they offer a *particular kind of lymphoid formation*, which shows at once its peculiar differences from other analogous structures. Among these peculiarities may be mentioned the presence, in their earlier stage, of one or more *giant cells*, the *entire want of vessels*, *the later solidification of the protoplasm of the giant cells*, that is, its *horny metamorphosis*, and the fibroid changes in the connective-tissue capsules.

The answer to the very important question of their histogeny, that is, which tissues and which cells give the incitation to the development of the tubercles, is, as you will discover from the great number of attempts which have been made at an explanation, very difficult to give. No place has been forgotten or escaped notice as a possible centre of growth. Their origin has been placed in the blood, in the walls of the capillary vessels, the sheaths of the smaller arteries, the lymphatic vessels, the connective-tissue layers of the vessels, and the contiguous parenchyma of the organ affected. The impression is always given, however, that those attempting the explanation have not

properly distinguished between the lymphoma and the growth surrounding it. Commonly the latter is taken for the tubercle, thus confounding one with the other, and rendering a decision much more difficult. The question evidently turns on the enigmatical *derivation* of the *giant cells*. E. Wagner has thought that we must admit that tubercles in the liver are produced by a proliferation of the nuclei of the liver cells. But the same investigator also admits their development from the intra-acinous tissues; and it is more than probable that the liver cells supply no material at all to the tubercle-lymphoma, but only take part in the new formative activity along with it. The same may be said of the walls of the capillaries (Colberg); their nuclei increase as though they had come within the reach of the irritation but without ever becoming tubercles, and their growth has much more to do with the formation of new capillaries.

Most commonly the cells already existing in the connective-tissue are regarded as the origin of the giant cells, and consequently of the future tubercle. Langham's view that masses of protoplasm are formed out of spindle-cells, which have no nuclei, and that these masses create nuclei in themselves, loses thus very much of its importance. Now, while in hypertrophy of normal lymphoid organs or in their increase by normal stimulants (supply of albumen, etc., received by the blood at the time of digestion), there is simply a production of cytoid bodies, yet as a result of abnormal irritation there is a production of giant cells. The endothelium of the lymph-sheaths of the finer arteries of the lymphatics (Rindfleisch, Klebs), and, perhaps also of the finer veins, are claimed, however, to possess the same power, viz. a production of giant cells.

An accordance between these latter opinions is possible if we remember that the juice-canals, that is, the spaces in which the connective-tissue corpuscles lie, are in communication with the canals of the lymphatic vessels, and probably with the lymph-sheaths of the arteries; and also that we

are entitled to admit the same ability to proliferate for the lymphatic endothelium as for the connective-tissue corpuscles. These *corpuscles would be then, not only the forerunners of fibrillary connective-tissue, but also of undeveloped endothelium.* The endothelium of the serous membranes and of the veins, whose openings lead into the spaces in the connective-tissue, would also have to be counted in the same category. We may say, then, that while in a physiological case of increased formative activity, only an increase of simple lymphoid cells takes place, in another case answering to a *particular irritation*, we find from the same elements a growth of giant cells; and these giant cells being once produced, all the other elements of a tubercle-lymphoma quickly follow.

My view demands, then, only the existence at the point of irritation, of a *pre-existing element from the series of connective-tissue corpuscles and endothelium*, chiefly from the endothelium of the lymphatics and serous membranes, from which the new lymphatic growth may develop and become further organized.

If you wish, like E. Wagner, to adopt the views in relation to tubercle-lymphoma proposed by Henle, according to which the development of the conglobate glands is to be traced back to a deposition in the original tissue of elements resembling lymph corpuscles, and according to which the stroma of the gland springs from a breaking up and fibrillation of the normal connective-tissue, even then it is not necessary to suppose in explanation an emigration of white corpuscles from the blood;* for certainly the theory of the formation of the *new elements from the connective-tissue corpuscles and endothelium* is quite as well authorized.

To the foundations which I have already given for my opinion, I will add that the small cells are not, like wandering

* For then we would have to go so far as to admit the origin of the giant cells also, from the lymph corpuscles, as Armaner-Hausen (Beiträge zur normal und pathology anatom. der Lymphdrüsen) has done.

cells, loosely packed, but are difficult to show in a preparation; and that they do not, like the wandering cells, undergo fatty degeneration, but are transformed into permament connective-tissue corpuscles.

It is certain that on the exuberant growth of these cells the fibrous system of the already existing connective tissue is broken up. *The reticulum of the lymphoma proper then is evidently a new formation, and seems to have its origin either in the secretion on their periphery by the giant and epithelial cells of a substance which subsequently grows hard and is analogous to connective substance, or by the peripheral substance of their protoplasm being transformed into reticulum by a hardening process.* This being the case, we can accept this fibrillation only for the outside layer of the tubercle. E. Wagner has observed also that this outside layer is separated from the lymphoma proper by a clear space, containing here and there growing endothelium cells, exactly as in normal lymphoid organs. As I hold it, however, to be inadmissible to affirm the existence in the connective-tissue capsule of an aggregation of white blood corpuscles which have emigrated from the blood, I must also certainly deny their existence in the lymphoma proper.

My view, founded on the fact that the connective-tissue corpuscles and the endothelium of the lymphatics belong histogenetically to the same category, presents now the advantage of making clear the supposition that the giant cells belong even to the increased connective-tissue corpuscles in the tubercle. The elements of the tubercle can be developed just as well from the connective-tissue cells (Langhams) as from the endothelium (Rindfleisch, Klebs) and at one time they take the form of cytoid corpuscles (young connective-tissue cells), at another that of giant cells, and again they occupy a middle position in the form of endothelial-like cells.

After all has been said, I cannot agree with the latest views promulgated by Schüppel, who conjectures that the formation of the tubercle takes place preferably within the

blood-vessels, and supposes mother cells in the blood, which are produced from coagulated fibrin, and are encapsulated by a growth of the endothelium of the vessels. In this case the giant cells must either be derived from the colorless blood corpuscles, which never, according to my knowledge, take on so enormous a proliferative energy; or one of the endothelial cells of the blood-vessels must have assumed this marked increase in size, a process which is not accepted by Schüppel himself, since he found the growing endothelium only encircling the mother cell; or finally, we must suppose the existence of circulating masses of protoplasm which stick in certain capillaries and remain there and grow to be giant cells. This latter is an hypothesis which at present would not be admissible on account of many counter facts, derived both from observation and theory. The growth of the neighboring endothelium synchronous with the formation of a tubercle, a growth which follows very quickly the formation of a blood clot, can very easily lead to the opinion that the tubercle is formed within the blood-vessels.

Schüppel founds his opinion on a single observation, an observation, moreover, which is capable of another interpretation.*

Through these deductions I have declared myself to be opposed to Waldenberg's theory, which he lays down in his otherwise classical work on tuberculosis, in which he declares that embolism of the capillaries by means of corpuscular elements is the seed of the growth of tubercles.

The fresh newly-formed tubercle of the smaller size is of a *soft gelatinous consistency, diaphanous, and has a color which approaches grey.* Since on account of their transparency the color of the organ shows through, they are

* The theory advanced by Hering (Studien über Tuberkulose, 1873, p. 105); that the giant cells are artificial products which are caused by the effects of the fluid used for hardening microscopic preparations, and that they represent the coagulated contents of the lymph-vessels which are cut, is easily disproved by fresh preparations.

commonly not very sharply defined, and thus escape the notice of the naked eye.

I have already mentioned that when they have reached their full size—since they have no vessels, we cannot expect them to be very large—they either undergo fatty degeneration, or become horn-like within, while externally they are encapsulated by hypertrophic connective tissue. In this stage the tubercle is *firm and dull, becomes opalescent*, takes on a *yellowish color*, and is rarely recognizable.

These changes always show a degeneration (*obsolescence*) of the tubercle; which is later changed into a fibroid nodule, and may be *entirely absorbed.*

The surrounding tissues which have participated in the growth play an important part in the degeneration. For the increased cells in the lymph-sheaths of the finest arteries, may so encircle and compress the arteries that they and the capillaries supplied by them are no longer pervious to the blood-stream. All the tissues immediately surrounding the tubercle, or the group of tubercles, are placed in a like condition; all together *fall into a state of anæmic-necrosis*, and as a result *undergo fatty degeneration.* In this way the tubercle appears to be much larger than it really is, that is, we say (but falsely) the tubercle is increased in size. The whole can break up together into a *cheesy mass*, and in rare cases, in consequence of a softening of the cheesy substance, a small *tuberculous cavity* may present itself. Externally the walls of the cavity contain lymphoma, but within are cheesy, while its contents consist of a soft yellowish pap-like substance, in the formation of which the whole group of tubercles and the whole surrounding growth, both of connective tissue and endothelium, as well as the parenchyma of the organ, have taken part, and are consequently destroyed. The formation of a cheesy cavity presupposes not merely that its size is the same as that of the cheesy mass—say, at least, a diameter of 4—6‴, but that the cells on its periphery are held fast and even eccentrically pulled upon (as in the walls of the bronchi in the formation of

bronchiectasis). If these conditions are wanting no cavity is found, but a *shrinking and formation of chalky concretions* takes place. These chalky concretions become surrounded by a fibrous capsule, the result of reaction, but may nevertheless be entirely absorbed.

We say, then, that a tubercle may be not only arrested in its development (obsolescence) and absorbed, but also that it may undergo cheesy degeneration and shrinking, and result in the formation of chalky concretions and even of cavities.

Properly speaking, however, the tubercle undergoes no other degeneration than obsolescence and absorption, the other changes belonging rather to the neighboring tissues implicated.

I beg you now to apply what has been said of tubercle in general to *tubercle in the lungs.* The tubercle occurs in the lungs, everywhere and only, where there exists connective tissue, lymphatics, and very fine arteries with lymph-sheaths. Agreeable to this we find them in the fibrillary layers of the mucous membrane of the larger bronchi, in the walls of the bronchioles, in the interlobular connective tissue, and even in the walls of the alveoli. Both the interspaces and the vessels of the connective tissue are incited by the formation of a tubercle to increased growth; and when the tubercle has its seat in the walls of the alveoli and interlobular tissues, the alveolar epithelium is also implicated.* The same is true of the pleural endothelium in case of a subpleural extension of the tubercles. If the tubercle is located in the wall of a bronchus, then there is a very active participation, particularly of the endothelium of the lymph-vessels and the epithelium of the mucous

* In relation to the alveolar epithelium there is more than a "taking part" in the tubercle formation; for they are themselves, as well as the endothelium belonging to the lymphatic system, the seat of tubercle formation and its pre-existing element, and the latter only spread further, from the inner alveolar wall into the lymphatics which have no walls.

membrane. This productive activity around the tubercular masses lasts only as long as the tubercle itself is fresh and active, and it abates and retrogrades when the development of the tubercle has ceased and its degeneration has begun.

Call to remembrance now, the description which I previously gave of genuine desquamative pneumonia, and which I declared occurred together with tuberculosis. You see, then, that an *acute miliary tuberculosis of the lungs*, considered from a clinical stand-point essentially as a local disease, is a *desquamative pneumonia*, which is only distinguished from the pure genuine form by the appearance of giant cells among the growing alveolar epithelium. The tubercles thus originally lie in the cavity of the alveoli, and the miliary tubercles are developed later in the swollen framework, which is filled with proliferated connective-tissue elements, as a result of local infection.

Now you are in a position to understand how miliary tubercles are formed, why, as a rule, they are diffused over both lungs from top to bottom, and why they are strewn through the parenchyma in innumerable quantities. You are acquainted with their seat, and the accompanying circumstances under which the growing alveolar epithelium appears to be modified by a much more brilliant quality and by a richer proliferation of their nuclei than in a pure desquamative pneumonia.

Miliary tuberculosis always occurs as an acute disease, and extends in its dessemination, not only from the apex downwards, showing as a result the youngest, smallest, softest grey and diaphanous tubercle in the lowest section of the lung, and those which are the largest, yellow, firm, and opaque, in the superior portions, but it also extends from the alveolar parenchyma towards the bronchioles. This peculiarity does not belong to tuberculosis alone, but is true of desquamative pneumonia, and in an equal degree, whether tubercles are developed in it or not. With the larger and yellow tubercles, as I have already

said, another very important element comes into play. You can easily comprehend that around the seat of the tubercle the parenchymatous swelling is greatest, and so, soon after the formation of the tubercle, an anæmic necrosis of the adjacent alveoli, that is, an acute cheesy lobular pneumonia, spreading by the addition of new points, is provoked, while a more wide-spread diffuse cheesy pneumonia seldom occurs.

What is generally considered to be a *large yellow tubercle of the lungs* shows under the microscope not only a lymphoma with central degeneration, but also a gangrenous and, soon to be cheesy, group of alveoli, with their desquaminated epithelium lying beside and between them; and this, in fact, makes up the greatest part of the yellow tubercle. Those who have overlooked the proper lymphoma in their investigation, cannot conveniently believe in the new growth theory. They imagine the miliary tubercle to be nothing more than lobular necrosis (Henle). While those who have not properly estimated the necrosis of the alveolar parenchyma, accredit to the tubercle alone the necrosis and cheesy degeneration, an interpretation which is given by most authors.

If the majority of the tubercles are very recent, without cheesy degeneration of the immediate surroundings, the name of acute miliary tuberculosis is most properly applied. If, on the other hand, the larger part have already become yellow and increased in size by the already mentioned cheesy changes in a surrounding group of alveoli, we must speak of a *subacute miliary tuberculosis*. What is called *chronic miliary tuberculosis* presents under any circumstances a very different appearance. If the point is strictly maintained, that it is the consequence of an acute process, the degeneration in the whole lung parenchyma, the punctiform cheesy degeneration, and the duration will show differences enough. If you have mastered the collective peculiarities of pure desquamative and cheesy pneumonia, as well as of tubercles, you will readily understand the

further course of miliary tuberculosis, and I shall not be obliged to explain it to you by a long repetition.

Miliary tubercles with their accompanying cheesy alveoli—this degenerative process begins afresh first in the apices of the lungs, and consequently is at this point always the most distinctly marked—occur gradually either in perfectly healthy lung tissue, or when the lung tissue is in a state of chronic fatty degeneration, and consequent richer myeline degeneration. The diffuse thickening and cirrhosis is developed from the apex on, but generally only in a very slight degree. The tubercle-lymphoma, often in the cheesy points but mostly in the cirrhotic, fibroid portions, either become quiescent or entirely disappear; so if there were no more recent tubercles to prove the presence of the characteristic process, it would certainly be very difficult to decide whether a given specimen was one which had originated from a peribronchitis and cheesy lobular pneumonia or from a diffuse simple desquamative pneumonia, or whether we had before us a specimen of true miliary tuberculosis.

Now, since obsolescence and absorption occur first in the tubercles of the alveolar parenchyma—and that they may be healed must be made most conspicuously prominent —and since these tubercles, which have their seat in the bronchial walls, are absorbed either later or not at all, a circumstance well worth remarking is brought to notice. The tubercles appear to advance by a local infection from the parenchyma in which they have disappeared further towards and into the bronchioles ; or in other words, the miliary tubercles which before stood thick in the alveolar parenchyma are now much more widely separated, their position corresponding to the bronchi. The reason that tubercles occurring in the bronchial walls are not easily absorbed is, that here the endothelium of the lymphatics is too deeply implicated and the cirrhotic thickening is too marked. These latter cases correspond to peribronchitis nodosa, which is in effect a chronic miliary tuberculosis.

As you have drawn your conclusion both from this and from the common account of the different phases of existence of tubercles, it will be clear to you that, in the lung itself, that is, in its parenchyma, there is in reality no other than an acute miliary tuberculosis which can spread in frequent acute relapses.

There frequently exists with the tuberculosis a secondary capillary bronchitis of a mild grade, but it is not invariable; it may be so slight that cough is entirely wanting. If it exists, however, and is accompanied by cough, with sputa, the character of these latter can be determined by a microscopical examination. The cyanosis which is sometimes observed in tuberculosis, belongs really to the desquamative pneumonia—the same is true of the consequent fatty degeneration which can attack all the organs and tissues of the body—events which are not without their influence on the termination of the patient's life.

LECTURE X.

Ætiology of Tuberculosis.

I ASK your attention to a brief *résumé* of the ætiology of tuberculosis.

Ever since I made my debut as a teacher in 1847, I have constantly entertained the idea that *miliary tuberculosis is a disease due to specific absorption and infection.*

Others may have held the same idea. By simply making assertions, however, nothing is gained; for those hypotheses can scarcely be considered as scientific, whose truth or falsity remains still to be proved by carefully observed facts. To have such ideas, to propose such hypotheses is certainly profitless; for they only obtrude themselves on every one who is working in the direction of scientific development. Only those assertions can be of use where truth is proved, and which, if they do not raise themselves to the level of a law, are at least combined with unavoidable conclusions drawn from an unbroken succession of proven facts.

Following out this idea as regards miliary tuberculosis, I have always striven to find for the new theory supports and foundations based upon anatomical facts, and ventured in 1856 the first publication of my discoveries.

Since that time the idea has been enlarged into a theory, particularly in consequence of the many successful cases of experimental inoculation which were published by Villemin (1865) and others. In 1856, and again in 1859, respectively nine and six years before Villemin's experiments, I made a number of inoculations on rabbits, but with insufficient results. I therefore contented myself with proofs taken

from the human cadaver, which settled in my mind the question of *tubercular infection in man* (however valuable the experiments on animals may have been), that being at the time the subject under discussion.

The infection theory has attracted many and weighty advocates—among them Niemeyer, Hoffmann, Waldenburg,* Bierner, Opholzer, Billroth, etc.; and the first of these it was who really infused life into the theory by bringing forward clinical examples which were supposed to include both tuberculosis and the diseased condition of the lungs which existed with it. There have, however, always been certain weighty authorities who have withheld their concurrence, and it would be quite in place to meet in some way their objections; for from their want of concurrence it seems evident that the theory is in some respects defective, and it lies now before me to remove as far as possible these defects. The theory rests upon the following propositions:

1st. *The foundation for the development of a miliary tuberculosis is a cheesy mass.* The taking up into the blood and lymphatics of the constituents of such a mass, causes the multiple development of tubercle (auto-infection). The *special irritant* then, which I mentioned in a previous lecture, is found—it is an *infection*. The first proof that cheesy centres form the points of departure, is the *almost constant presence somewhere in the body of one or more such centres or masses.* The concurrence of tuberculosis and yellow masses, the size of a bean or walnut—formerly called crude tubercle—was long ago recognized by Laennec, Rokitansky, and others. But Rokitansky did not realize it to be genetic, and Laennec† considered only the softened masses as active, and deceived himself as to their true nature. Virchow,‡ who is not to be taken as altogether favoring the theory, concedes that the question may well be raised, whether in general an eruption of tubercles does

* Waldenburg only accepts the theory in part.

† Diseases of the Lungs, I. p. 438 and 512.

‡ Geschwülste, II. p. 724.

occur without the pre-existence of cheesy or (exclusively in Laennec's sense), softened "mother nodes;" and that the answer must be made that it is certainly a very rare occurrence; for if only a thorough examination is made, an old cheesy node is found in every case.

I have, however, never given it as my opinion that they exist in every case, and am at a loss to understand how I am accredited * with having proclaimed their invariable occurrence; the "*always*" has slipped in through carelessness. On the contrary, in the history of eighty-four cases,† I have shown that in seven cases, that is, in 8 per cent., no cheesy nodes would be discovered. Later in the history, three hundred cases, contrary to my expectation the proportion was still more unfavorable, viz. 10 per cent. Still the number of cases in which cheesy nodes were to be found remained always so surprisingly large that they still counted for the foundation of the theory.

2d. Not less important is the proposition that *the cheesy nodes which lead to the miliary tubercle can be referred back for their origin to a previously existing inflammatory process.* The material for the infection is commonly furnished by cheesy degenerated pus in the connective tissue; an example of which I showed‡ in a psoas abscess; also by cheesy nodes in lymphatic glands previously the seat of inflammatory irritation (scrofulous); by both the external and internal cheesy degeneration of bones, particularly in their articular ends and in the vertebræ; by cheesy degeneration in the testicle, in the mucous membrane of the uterus and tubes, and of the intestinal canal, and in the brain; also by cheesy degenerated purulent exudation on the serous membranes, centres of cheesy pneumonia, the walls of the resulting cavities and small infarctions from emboli and more seldom cheesy degenerated peribronchitis.

* Virchow, l. c.; Otto Niemeyer, l. c. p. 495; Schüppel, Tuberculosis of the Lymph Glands, p. 131.

† Wiener Wochenschr. 1859, No. 13.

‡ Wiener Wochenschr. loc. cit.

Most of the experiments in inoculation agree with this, and where material has been successfully employed which was not the product of inflammation, it has acted by creating first an inflammation at the point of inoculation, and then the products of this inflammation, by undergoing cheesy degeneration, have furnished material for an auto-inoculation (Waldenburg). If the cheesy nodes must have had an inflammatory derivation, then the negative results from the use of other materials are accounted for; and we are forced still more to perceive, in the *infectious material of the cheesy centres, from which tuberculosis results, something specific.* We are still more strongly urged to the adoption of this idea, by seeing soon after an inoculation with cancerous material, the advent of a miliary carcinoma, a disease greatly resembling miliary tuberculosis, but easily distinguished from it by its anatomical peculiarities. Although we see nothing specific in the inflammation, nor even in the cheesy degeneration of its products, still the effect, the generation of miliary tubercles, formations which can be distinguished from all other new growths by perfectly distinct anatomico-histological characteristics, has something so peculiar and specific about it that we are logically forced to call the cause, the tubercular material which is generated by the cheesy degeneration, a specific infectious substance.*

3d. In order that the supposed absorption and infection may go on easily and without any hindrance, it is necessary to admit the next proposition, as follows: *the cheesy nodes are not entirely encapsulated,* that is, they are *not enclosed on all sides by firm fibroid tissue.*

This is a point which has been very often overlooked; for there may be cheesy nodes which do not lead to tuberculosis, and this fact may certainly be taken as evidence against the correctness of the infection theory. It is averred

* I will discuss later the idea advanced by G. Schüppel that the inflammation, which leads to cheesy degeneration and tuberculosis, must be in itself peculiar (scrofulous).

in reply that there are also ichorous collections which do not lead to pyæmia—a fact, but at the same time a very weak attempt at explanation. It would be better to adopt O. Niemeyer's idea that the cheesy masses by progressive changes lose their pernicious properties. Much the strongest argument, however, is that the nodes are encapsulated, and although the hindrance offered to absorption by cirrhosis and by the limiting cicatrization is not absolute, it is still very great. The pus which is derived from the surface of a mucous membrane and is retained in the mucous membrane canals, is not in a condition to generate infection, even where it has undergone complete cheesy degeneration. Catarrhal pneumonia and bronchitis, croupous pneumonia and bronchitis, are scarcely able to awaken a miliary tuberculosis. On the other hand, the cheesy masses in the connective tissue, derived from the cheesy products of inflammation, and which extend to a certain degree by their prolongation into the lymphatics and blood-vessels, may certainly, before the connective tissue has undergone cicatricial hypertrophy, give occasion to infection.

4th. An important means of proving that miliary tuberculosis has its origin in absorption is the *infection of the adjacent tissues.* This adjacent infection probably occurs only through the lymphatics or juice canals; for the tubercles always appear particularly thick in the immediate neighborhood of the centre of infection, and spread at intervals eccentrically from this point. The more recent the infection, the more do the freshest and smallest granulations immediately adjoin the point of departure: the longer it has existed, the more is the cheesy centre surrounded by the oldest and largest tubercles, which have already become yellow, and which by absorbing the smaller granulations appear to be more widely separated from one another; while on the contrary those recent and grey and which are closely placed are more widely removed from the centre of infection. If this centre is in the lungs it is generally in the apices and the contiguous infection corresponds.

I offer as a very striking example of adjacent infection the case of a girl eight years of age, in whom, in consequence of caries of the lower dorsal vertebra resulting in cheesy degeneration, and following previously existing pleural adhesions, an extensive ulceration had eaten into the parenchyma of the lower lobe of the right lung. Miliary tubercles were developed only in this lung, and were thickest on the circumference of the ulcer, extending, however, eccentrically from below upwards, a manner the reverse of what is ordinarily met with in the lungs.

Analogous formations of miliary tuberculosis are found co-existing with cheesy masses in the brain, but are limited to the pia mater; with intestinal ulceration they are found only on the external intestinal covering. I once saw a local instead of a general infection take place as the result of a cheesy thickened psoas abcess on the right side; the adjacent cæcum, and this alone, was crowded all over without and through and through even to the mucous membrane with small groups of miliary tubercle. Zenker* declares that there are no more important grounds for referring the origin of miliary tubercles to cheesy deposits, than the occurrence of cases of local circumscribed miliary tuberculosis. Similarly, pustules are noticed around furuncles and suppurating blisters, and a measle-like exanthema around a vaccination pustule.

The adjacent infection is generally comparatively slow in its development, and the name "chronic tuberculosis" could be very conveniently applied to it. It travels along the bronchial walls from the lung parenchyma, and causes a grouped or discrete chronic miliary tuberculosis.

5th. As in certain cases there is a local infection of the adjacent tissues, so in others we meet with a *general infection*, which may be taken as a further argument for the truth of our theory. To account for this general infection we must suppose an *infection of the blood.*

The tubercles seen in this form are nearly equal in age,

* Sitz. Ber. der Erlang. phys. med. Societ.

fresh grey, diaphanous, and very small, and are found in the tissues of *nearly every organ in the body:* the invasion is acute, and occurs entirely without reference to the position of the centre of infection, though there is but one such centre in the body. The most commonly chosen situations for the eruption of tubercles of the lungs are the basilar portions of the pia mater, the mucous membranes of the air passages and digestive tract, the serous membranes, the muscles of the heart, the liver, spleen, kidneys, etc. The tubercles developed in the choroid coat of the eye have a quite particular significance.

The age at which a general infection is most common is before the twenty-fourth year, that is, while the body is growing, and the production of blood and lymph most active, this being especially a predisposing cause.

But we must examine the clinical aspects of this disease as well as its pathological anatomy. It was this general tuberculosis in particular which stood in the way of the acceptance of the infection theory; for in ten per cent. of the cases is it not said to have occurred independently of any cheesy spots? I have already remarked that a negative result on making a section does not prove the non-existence of an infectious centre; for, according to an analogy with other infectious materials, only a minimum quantity ought to be required in order to create the disease. It cannot be questioned that the infectious material does not necessarily need to be pressed together in a mass, or that it can be produced and developed to a certain degree out of the products of retrograde metamorphosis, which are dispersed throughout the body as a result of general changes of nutrition. In reality, four-fifths of the cases in which no cheesy degeneration was found can be explained in this way; for, singularly enough, three-fifths were between the ages of fifty and sixty, and in the fourth one-fifth severe diseases had preceded (3 puerperal fever, 2 typhoid, 1 morbus Brightii); so that only one-fifth, or 6 out of 300 cases, that is, two per cent., remain unexplained. These cases would agree

with Ditterich's infection theory, so far as relates to the definition of the cause, but not as regards the result. Hoffmann,* has thoroughly appreciated this difference when he says, "Ditterich has nowhere mentioned the appearance of tubercles, but lays the principal weight on the inflammatory diseases." Here may be classified also the cases which Waldenburg † has collected, where tuberculosis has occurred after perforating ulcer of the stomach, diabetes, Banting cure, and suppression of the usual secretions (chronic exanthemata, ulcers, hemorrhages).

6th. Another important argument is the *situation of the miliary tubercles in the connective-tissue containing the lymphatics of the organ, and the analogy in their histological formations with normal lymphoid organs;* an analogy which extends even to the *physiological conditions* which they call into life, though not at all to the functional.

Tubercles are recognized as being pathological and different from the normal lymphoid organs, by their occurrence in unusual places (heteroplastic growths), and by the inflammatory irritation which they occasion in the adjacent tissues, by their occurring irregularly and in countless numbers, by their quickly becoming firm through horny degeneration of their cells, by their lack of vessels (Schüppel), and by their transitory existence, that is, their tendency to speedy degeneration and fibroid shrinking. If we accept the generally received view, that the lymphoid organs serve for the formation of the blood, and if we can also admit that the fluctuations in their presence, that is, their appearance and increase through an active internal cell formation, and their disappearance, have direct connection with the co-existing condition of the blood,‡ then we can affirm the

* Deutsches Archiv f. klin. Med. III. 1, p. 122.

† Loc. cit. 508, 509.

‡ As examples of the different conditions of the blood, we may mention those produced physiologically by a state of hunger or thirst also the pathological conditions seen in leucæmia, scrofula, typhoid etc.

same of tuberculosis; and say that the occurrence of a tubercle-lymphoma must be considered as the necessary result of a certain blood condition, or, more comprehensively expressed, of a certain condition of the tissue juices created by the absorption and irritation of cheesy material. But this material cannot be absorbed and circulated without some coincident changes in the lymph and blood vessels, for it will by its seed-like action arouse the formative activity of the connective-tissue corpuscles and the endothelium of the lymphatics, and manifest itself by the production and proliferation of cells and nuclei. Tubercles then may originate either in the circumference of the centre of infection, in a distant organ, or as a general disease.

Considered in this way, tubercles are not a simple sediment deposited by a dyscrasic blood, for there is connected with their origin a perfectly distinct incitation, and a marked activity in organization; neither are they compounded according to a chemical formula by retrograde metamorphosis of an exudation, since they act from the very start as newly formed tissues, and then decomposition takes place only when their development is completed. Again, it is not possible that they can be simply emigrated white blood corpuscles deposited in little groups, that is, secondary, derived from primary centres of inflammation, because as a result of such a process we would have, not a lymphoma, but a formation of pus; and for the purulent collections we must accept the existence of scattered punctiform stations of emigration, and the primary centres of infection must be quite fresh at the time of the production of the secondary, just as in pyæmia. If the tubercles really are formed from lymph and blood corpuscles, then ground is gained for the theory of embolism, which has been put forward by some authors in opposition to the infection theory (Parrum, Wyss, Waldenburg). But the emboli are only conjectural, having never been actually seen; and if the carrying away of corpuscular elements from the cheesy centre has been really proved, then an embolus consisting

of a mass of degenerated cheesy tissue must represent a tubercle; but if it has not been proved, then the development of the lymphoma can only be explained by the action of molecules brought into direct contact, that is, infection. Thrombosis of the lymphatics would hardly occur to any one wishing to adopt the mechanical absorption theory, even where a growth of the endothelium and a formation of tubercle might give some support to such an idea.

Perhaps we may be able to reconcile the embolism theory and my infection theory in the following way: If we consider the infectious material, aside from its chemical nature, to be similar to semen, that is, as a fluid containing little bodies which resemble spermatozoa and have the power of spontaneous movement, then the corpuscles are not bodies capable of becoming emboli, but are simply molecular bacteria, such as I mentioned previously, and which are to be met with in cheesy masses. These may indeed reconcile the theory of the direct action by contact of the specific material derived from the cheesy masses with the theory of its catalytic action. We certainly could not consider these corpuscles as loosened cheesy particles of a size to stick and remain in the blood and lymph capillaries, neither as amoeboid cells, since in the cheesy masses, besides detritus, there are only dead cellular elements and shrunken nuclei, nor still less as the rudiments of such cells, but we are forced to consider them as bacteria. The mycelium filaments, for for these at least a spontaneous movement is proved, are brought in contact with the connective-tissue corpuscles and endothelium by the current of the tissue juices they bore into the protoplasm of the cells, and increase up to a certain point at its expense, and may at the same time give the impulse to a proliferation of the nuclei. The result of this impulse may be a giant cell which follows the other elements of the tubercle.

The *granules within the giant cells are then not simply protein and fat molecules, but are in great part round and rod-like bacteria* (see note, p. 99). If the increase in the bacteria

is not very great, there may at least be a formation of cells resembling epithelium or a congregation of cytoid bodies as in normal lymph-follicles. If the bacteria perhaps assist in producing the specific material in the cheesy masses, they are still more likely to be the vehicles of the infection, and thus there is a possibility of their being a fungoid contagion for true tuberculosis.

7th. Tuberculosis is not an isolated instance of the production of disease by infection; we find analogies in other diseased conditions, in *miliary carcinoma*, for example. This disease always owes its origin to an infection from a primary cancer, multiple depositions, either in the immediate neighborhood, or throughout the whole body, showing themselves as a result. Tuberculosis and carcinoma are distinguished, however, by the entirely different nature of the cells which go to make up the cancerous lymphoma, and by their consequently presupposing a distinct infection for each—one, only a cheesy degeneration of the products of inflammation, and the other a material capable of originating a cancer. Miliary carcinoma occurs very rarely compared with the frequency of secondary cancers, which are caused by a mechanical absorption of living cellular elements. Secondary cancers always follow exactly the structure of the original growth, as is shown in pigmented cancer, malignant osteoid, and in gelatinous and flat or cylindrical epithelial cancers; and must not be confounded with cancerous lymphoma. We must place in the same category secondary sarcoma, enchondroma, fibroma, and osteoma, which have nothing in common with the peculiar unmistakable structure of a lymphoma.

The elements which make a cancer infectious are those which are derived from a degeneration peculiar to itself. It is not simply the original structure which makes a new growth cancerous, but it is the degeneration which it is capable of undergoing. Fibroma, osteoma, enchondroma, sarcoma, and particularly adenoma, become malignant through degeneration: what stamps them as cancerous are the pro-

ducts which come from degeneration, and which lead to absorption and infection. The proposition which applies to tuberculosis and pyæmia, that so long as an inflammation is active, so long its products cannot become putrid or undergo cheesy degeneration, thus rendering absorption and infection impossible, applies equally well to new formations; so that we may say, as long as any neoplasm grows, it remains non-malignant, that is, a miliary carcinoma is not yet to be feared.*

8th. In addition to these points, we must regard as favorable to the infection theory the fact that tuberculosis *can be conveyed from one human being to another.* I do not mean the inheritance by a child from its parents, but the immediate communication through contagion, both that which takes place from one part of the body to another, and from one individual to another. This is shown in the examples adduced by Rindfleisch (p. 349), where tuberculosis was conveyed over a limited space from the pleura pulmonalis to the pleura costalis; also in a case which I met with where a true tubercular ulcer on the edge of the tongue communicated itself to the adjacent mucous membrane of the cheek.

So also additions are being constantly made to the cases where the contagion has been conveyed from a husband to the wife, or *vice versa*, though this might perhaps be explained on the same ground as the inheritance from parents. This also agrees with the well-known contagiousness of glanders, which is only a very pronounced form of miliary tuberculosis, and with the course of general syphilis. This latter is an analogy which we must not overlook, since the histological characteristics of the syphiloma are so similar to those of the lymphoma as to be readily confounded with them.

9th. Still another fact which tends to support my theory is that *tuberculosis, as a rule, does not co-exist with any other disease derived from infection.* Rokitansky was the

* The hint which I have here thrown out in regard to a definition for cancer, I hope to be able to follow out on some other occasion.

first to call our attention to what we might call this active property of exclusion; an exclusion, however, which is not to be considered as absolute: he accredited it to all forms of cancer and to typhoid fever. I may add, by way of parenthesis, that Rokitansky did not propose this for the purpose of separating true tuberculosis from parenchymatous inflammation of the lungs. As far as my experience goes, I can affirm the same rule to hold good in pyæmia; and have long since called attention to the very rare occurrence of tuberculosis simultaneously with chronic purulent discharges.* Bilroth and Mengel † have corroborated this latter conclusion, basing their assertions on a very great number of cases. Such negative demonstrations only prove, however, that where there is no cheesy mass we are not to expect tuberculosis.

10th. As the final, but at the same time the most weighty argument to prove that the origin of tuberculosis lies in absorption and infection, I must again mention the *results of inoculation, proving as they do that tuberculosis can be conveyed to animals.* This result, obtained by experimentation, is one of the brightest triumphs which has been achieved by experimental pathology.

* Bei über 280 Sekt. etc. p. 111.

† Langenbeck's Archiv f. Chirurgie, xii. p. 365.

LECTURE XI.

Tubercular Pneumonia.

I DO not dare to leave the subject of tuberculosis without noticing the following very important questions: First, *is tuberculosis a product of inflammation?* and second, *what is to be understood by tubercular inflammation?* As regards the first question my position is peculiar. It is an indisputable fact that acute tuberculosis and inflammation do co-exist, the development of tubercles in the lungs being accompanied by a desquamative pneumonia, as I have already mentioned; but that the tubercles are either the cause or the result of the inflammation, I am not so willing to admit.

We often hear the opinion advanced that the tubercles irritate the tissue surrounding them, and thus tend to excite an inflammation—an opinion hardly more advanced than the old primitive idea, according to which, after the occurrence of corruption in the blood there takes place a simple and critically favorable throwing off and deposition of the materies morbi in the form and shape of tubercles; and these tubercles thus deposited become of themselves in turn injurious, and, like any foreign body, excite inflammation. With the knowledge that the development of miliary tubercles shows a rapid formation of tissue, in which all the surrounding tissues for a certain distance take part, and that this process, being acute, seems to come under the head of inflammatory phenomena, all attempts to separate them in respect to the time of their occurrence, regarding the one as the cause, the other the effect, ceased. They are in reality synchronous, both the effects of the same cause.

Acute miliary tuberculosis, therefore, considered merely in its local aspect, *is an inflammation accompanied by a development of tubercles.*

The process which takes place in *general* miliary tuberculosis, however, we must not so lightly call inflammatory; following thus the example of those who have even gone so far as to attempt to include under the term "inflammation" other general disturbances of nutrition, general acute fatty degeneration, for example. The word inflammation implies the occurrence of an uninterrupted infiltration of a tissue with the products of inflammation as far as the process extends. Tubercles, though sometimes discrete, again may lie so close together, even compressed and united in a mass, that we may speak of a *tubercular infiltration.* By admitting the possibility of a tubercular infiltration we do not give up the idea that tubercles always, under any circumstances, as the name demands, preserve their simple and individual forms, even though they may lie very close together. *Strictly speaking, there is no such thing as a diffuse tubercle.* What is here really of importance is quite another thing.

I must ask you to recall what, when speaking of cheesy pneumonia, I gave as the most important cause for the occurrence of necrosis in desquamative pneumonia. "A development of cells with small shining proliferating nuclei, accompanying the finest arterial twigs externally, seated in their adventitia, and causing this to swell up into lumps by reason of its irregular force, sometimes losing itself diffusely, and again rising up diffusely." I mentioned at that time also that this cell infiltration accompanied every desquamative pneumonia, and gave it as a mark of distinction from the consecutive form; also that it generally occurred in such considerable quantities that cheesy pneumonia is much more commonly met with than pure desquamative pneumonia. If we now seek for the point of origin of this exuberant cell-growth, we find it partly in the cellular elements of the connective tissue, as in pure desquamative pneumonia,

and partly in the endothelium of the lymphatics, particularly those which exist in the adventitia of the arteries, as in cheesy pneumonia; and also that it may even originate in the alveolar epithelium. The growth in both situations shows the diffuseness of the distribution: if it occurs irregularly, it shows a peculiar tendency, in one place to an hypertrophy of the connective tissue (cirrhosis), and in another to cheesy degeneration of the epithelium of the lymphatics and alveoli which are shut in by hypertrophied connective tissue.

If we were quite wrong when we formerly called everything which was cheesy, tubercle, and consequently called cheesy pneumonia tubercular, we at least thought that we had gained the right, after the discovery of this cellular infiltration, to speak of tubercular infiltration, of tubercular inflammation, and of diffuse lymphoid adenoma; and still more certain were we when we learned to recognize the processes and radii of miliary tubercles, which we wrongly supposed to have exactly the same origin and termination, viz. growth of endothelium, with cheesy degeneration of the lymphatics and hypertrophy of the encircling connective-tissue.

According to our present knowledge concerning the nature of tubercles, the question whether an infiltration of cells is tubercular or not cannot be decided in the affirmative, simply because the cell development, though generally regular, is in some places collected in little masses which give us the macroscopic appearance of tubercles. Nothing but the recognition of the structure of a true lymphoma can mark the infiltration as specific and prove it to be tubercular; and in fact *tubercle-lymphoma may be discovered in a cellular infiltration which is undergoing cheesy degeneration.*

In choosing the best definition for tubercular inflammation we have to decide between the four following: 1st. *Any inflammation resulting in cheesy degeneration which induces a tubercular infection might be considered as a*

tubercular inflammation, for it is an inflammation which furnishes the germs for a tuberculosis, even if at the *point of inflammation itself tubercles do not make their appearance.* Such a definition could only be given as it were *post festum;* for the suspicion of an ability for tubercular infection could not be cast upon the inflammation and its products alone, for we would only know of it when the fact of the infection, shown by a formation of miliary tubercles, lay already before us. No one, not even Schüppel, who comes nearest to it, would be willing to advance such an idea.

2d. That inflammation only can be called tubercular *which has provoked an eruption of tubercles immediately by the side of and within the cheesy masses.* Such an inflammation, from the very conditions, must long before have ceased; and by the side of the cheesy masses which remain as its results, the tubercles would appear as fresh as possible, and would indisputably be set down as being *to a certain degree secondary and of later occurrence*—in fact, as the *products of local infection.* Such cases are very common. They are to be distinguished from those coming under the first head, not by their nature but quantitively and according to the seat of the process. In these cases the infection is only local, while in the former it was general, the immediate surrounding of the centre of infection even escaping altogether. The cheesy masses and the preceding inflammation, however, remain in both cases the same. The fact that where a local infection has already arisen both around and in the centre, a general eruption of miliary tubercles may ensue, does not need to be more than mentioned. These were without doubt the cases which led Schüppel to the conclusion that a general infection proceeds from an organ previously affected with tubercles.

3d. We may call *any inflammatory process in an organ a tubercular inflammation, during the continuance of which,* as a result of previous infection, *tubercle lymphoma are developed.* The formations of the tubercles and the inflammation are in this case coincident; fresh tubercles are

formed not only by the side of the cheesy remains, but also by the side of and within the compass of the active inflammation. A retrogressive inflammation with degenerating products may include also retrograded obsolescent tubercles. A careful examination would allow of an easy differentiation between cases 2 and 3.

According to this, a desquamative pneumonia combined with a development of miliary tubercle would be a tubercular pneumonia (acute miliary tuberculosis of the lungs). This definition is certainly very seductive, but it presupposes somewhere in the body a centre of infection, possibly, even generally, quite separated from the inflamed organ. Neither does the affection itself remain confined to the lungs, but endangers more or less all the organs and tissues of the body. Strictly speaking, this does not harmonize with the idea of the inflammation. As I said at the beginning the best appellation for such a process would be "inflammation accompanied by a formation of tubercles."

4th. We are thus forced to the adoption of the last of the four definitions, which is, that *only that inflammation can properly be called tubercular, which not simply by chance, but by its own inherent qualities possesses the property of necessitating a production of tubercle-lymphoma and of having as in the third case the appearance of the lymphoma co-incident with the inflammation, and of having them confined to the tissues in which the inflammation takes place.* This definition agrees equally well with our conceptions both of the inflammation and of the tubercles; and applying it, we can no longer have any doubt as to which of the previously mentioned inflammations of the lungs deserves the name of tubercular. *Cheesy pneumonia*, with its exuberant growth of cells in the endothelium of the lymphatics, not only conditionally, but by its very nature, possesses the necessary conditions for the future production of tubercles, and when they do occur the lymphoma remain confined to the inflamed lung; it can then claim as a synonymous title the name *tubercular pneumonia* (*Laennec's* tubercular infiltration).

The essential *pathological* mark of distinction would be the *absence* of any *centre of infection;* for tubercular pneumonia is to a certain extent primary, while the *acute miliary tuberculosis* is to the same extent *secondary*, that is, the *result of infection.* Anatomically speaking, the difference does not lie in the diffuse tubercles as opposed to the discrete miliary tubercles, for the structure of the lymphoma is essentially the same in tubercular inflammation as in miliary tuberculosis, except that the tubercle resembling the normal lymph-follicles preponderate over those containing giant cells; neither does it lie in the close packing of the tubercles (infiltration), since in miliary tuberculosis they may be just as closely packed. It consists partly in the continuous confinement of the process to the involved organ or tissue, and partly in the diffuse cell infiltration which in miliary tuberculosis exists only in the immediate neighborhood of each single lymphoma as a local process. In tubercular pneumonia the cell infiltration is the most important part of the process, and assists in an uninterrupted reciprocal binding together of the lymphoma. If we attach the chief importance to this cell infiltration, "the inflammation accompanied by a formation of tubercles," the "miliary tuberculosis anatomically considered" would be exactly like the tubercular inflammation, and the difference would be simply and solely the absence or presence of a centre of infection.

If now we wish to reconcile these facts, we must admit that there are two methods of occurrence for tubercle-lymphoma, the *secondary miliary tuberculosis due to infection, and the primary tubercular inflammation.* On account of the power which the infection theory exercises, it is difficult for us to imagine a primary formation of tubercles. We can, however, find many anatomical analogies. For example, there are two forms of spleen in leucæmia. In one, the pulp being at the same time increased, the formation of lymphoma is limited to enlargement of the existing and creating of new malpighian bodies, while in the other the

cell growth is developed all through the spleen in the coats of the arteries; that is, the disease is diffuse. In the same way there are two sorts of syphilitic liver; the one presents nodular syphiloma with obliteration of the larger branches of the portal viens and cicatricial contraction; the other is distinguished by a diffuse proliferation of small cells accompanying the branches of the portal veins deep into the parenchyma of the organ. Again, we have two forms of amyloid spleen, the so-called sago spleen and the diffuse amyloid spleen; and even under normal conditions there is by the side of and proceeding from the lymphoid organs a diffuse development of cells (Kölliker, Kyber). In the same way we have supposed an interminable extension of cell infiltration beside the formation of tubercles, along the pulmonary arteries, and beside the lymphatics, along the interstitial connective tissue, in the pleura. If we deny the truth of the infection theory, as we may do, not only in those cases where no cheesy centres could be found, but even in those which testified to the truth of the theory, and if in so doing we still preserve the necessary impartiality and love of truth, we shall be easily satisfied with the explanation offered in this twofold manner of occurrence. We can then classify all cases of acute miliary tuberculosis without recognizable centres of infection, under the head of tubercular inflammation. Our pathology is not, however, fully satisfied, and we still have to find a mode to account for the occurrence of a primary tuberculosis.

Tubercular inflammation is characterized by very distinct peculiarities: it is not distinguished simply by a simultaneous development of tubercle-lymphoma, for this would apply equally well to an acute miliary tuberculosis, but rather by the proliferation of the endothelium of the intermediary lymphatics, and by the participation of the connective tissue together with embryonal new formation of this tissue and its vessels. As a result of this process there follows, moreover, immediately capillary anæmia and necrosis, cheesy degeneration, the formation of ulcers and

cicatrices, perhaps also collateral hyperæmia and secondary tuberculosis from local infection.

The question must obtrude itself, on what supposition can we explain the occurrence of an inflammation with such peculiar characteristics?

According to our present knowledge we can, unfortunately, only give an answer in the highest degree indefinite, as when we point out a supposed peculiarity of constitution. I have already in a previous lecture (V.) remarked that general desquamative pneumonia is the localized expression of a general disease. In order to make the same expression true of cheesy and tubercular pneumonia it must be very much modified, since that form of pneumonia is only a higher grade of pure genuine desquamative pneumonia. As regards the expression general disease, we know that it is in the fullest sense of the word *constitutional*, that is, it is interwoven with the whole organization of the body, both as regards its structure, nutrition, and irritability, that it is very commonly *hereditary*, and that it is more commonly transmitted from the father than from the mother to the children and grand-children.* In many cases the consti-

* We have all met with examples of hereditary transmission. There are, however, remarkable cases in which the parents have reached a good old age, but the children have been phthisical. I know of one family where both father and mother reached the age of seventy, while eight of their nine children perished from phthisis. The mother had a cirrhotic mass with chalky deposits in the apex of one lung.

In another family, the father, a gouty but otherwise strong man, had never in his whole life any chest symptoms, but the children by both his wives, who neither of them had phthisis, were most of them attacked. Out of nine, seven died suddenly, just as they had grown up, of cheesy pneumonia. One of the two living sons, and the eldest of all, has a cavity with cirrhotic surroundings in the apex of his lungs, and the other is entirely healthy. The inheritance seems here most certainly to have been derived from the father, for one of his brothers died of phthisis. In a third family there were three brothers between sixty-five and seventy-five years of age; one died of cancer of the rectum, the two others are living and have always enjoyed good health. One

tution does not show any striking signs which declare the general character of the disease; in others, however, it is declared by a feeble build, flabby muscles, narrow thorax, a small volume of motor force, increased nervous irritability, and particularly by a weak power of resistance in the organism, which to the slightest provocation responds with a deep-seated inflammatory disturbance. The respiratory organs are perfected in their development during growth, and are at this time the most irritable, vulnerable, and easily subjected to inflammatory disturbances of any organs in the body.

I might in this connection use with some changes the words of John Simon (Lecture on General Pathology) where he says, "A child repeats exactly, even to its final development, the development of its father. It becomes corpulent or gray at the same age; similarly either the top of the head, the temples, or the forehead are the first parts affected by the greyness; it also loses its teeth at the same age, the pulse is the same, and there are the same habits as regards sleeping, waking, etc. The types of defective development are transferred in the same way. The blood is, as it were, an extract of the whole organization both of the developmental and degenerative metamorphosis; and in the different periods of life, in the most widely separated organs, certain specific materials will prove this peculiarity of its development. The tendency to cheesy pneumonia is then an hereditary developmental disease of the blood, of the lymph, in a word, of the whole organization. The child inherits a disposition to the formation of such a peculiar blood that, as a part of the result, either from an extraordinary or a common irritation, a cheesy pneumonia is produced. Cheesy or tubercular pneumonia forms then a portion of the developmental type of the child."

That this is a general disease we learn from the occurrence,

has already lost four, another three, and the third one child from phthisis; and suspicion is already directed against some others of the children of each on account of their poor constitution. The grandfather died of phthisis.

in the commencement of the inflammatory process, which is the acute beginning of the pure or cheesy pneumonia, of *symptoms resembling typhoid*, as I have previously stated and have since repeatedly seen corroborated. This resemblance is not simply seen at the sick-bed, but is confirmed by the anatomical appearances; for we see an acute swelling of the bronchial and mesenteric glands, and of the solitary and aggregated glands of the mucous membrane of the intestine, which, if the course is chronic, leads to ulceration. We have further proofs of its being general in the frequent *acute relapses*, so that we can only be certain of the extinction of the abnormal constitution when the inflammatory recurrences cease; and again in its beginning in the apices of the lungs and quickly or slowly extending * downwards; also by its becoming indefinitely chronic, lasting until it finally kills.

If now tubercular inflammation is founded on a general affection, it may be found both primary and isolated, not only in the lungs, but in other places in the body; indeed there is scarcely an organ or tissue in which it has not been observed. Besides the lungs, whose proneness to tubercular inflammation is easily explained by the presence of the alveolar epithelium belonging to the lymphatic system—if we should imagine the lungs and their alveoli to be spread out flat, their condition would be made to resemble a serous membrane—there are the serous membranes, which are particularly liable to attacks of tubercular inflammation, the inflammation even taking on a subacute form. We meet also with primary tubercular pleuritis, pericarditis, peritonitis, periorchitis, arthritis, and meningitis. Primary tubercular inflammation is of much rarer occurrence in the mucous membranes, and has a much more chronic course; as in the urinary tract (pelvis and tubular substance of the kidneys, ureters, bladder, and urethra), in the fundus of the uterus, the tubes, the larger bronchi, trachea, and larynx, †

* Loc. cit. 280 Autopsies, etc., p. 65.

† Primary tubercular laryngitis is of especial interest, as it is often the forerunner of peribronchitis nodosa complicated by lobular

the buccal mucous membrane, and the whole intestinal canal. In the parenchyma of the organs it is not of such rare occurrence; the cheesy nodules found in the brain depend on a tubercular encephalitis (proliferation of the endothelium and perithelium of the arterial sheaths, new formations of embryonal connective tissue and softing of the brain substance between), and they enlarge by the addition of new layers, through eccentric extension. We meet also with a tubercular inflammation of the lymph glands, testicles, spleen, and liver. We also see it attacking the periosteum, the porous portions of the bones, the inner surface of the dura mater, and the connective tissue generally. Tubercular inflammation of organs is very favorable to the occurrence of secondary infection. It does not of course belong to this occasion to discuss particularly tubercular inflammation in all these different organs and tissues, and to conclusively prove the reality of its occurrence in each.

The histogenesis of tubercular inflammation can be most easily followed out on the serous membranes, which are very suitable objects for study as compared with other membranes, and particularly with the lungs. This is quite comprehensible when we remember the relationship of the endothelium, with the connective-tissue corpuscles on the one side, and with the endothelium of the lymphatics on the other. On the serous membranes tubercular inflammation shows a formation of embryonal connective tissue (desmoid fibrinous formations, production of granulation tissue) containing delicate was capillaries whose loops are connected in a network and which exude either semen or blood (hæmorrhagic exudation). We may recognize in this embryonal cellular tissue, microscopic lymphoma in the very

disease of the lung parenchyma, and is hence classed as of the same importance as the latter. It is clearly all the same whether the general disease develops, as it generally does, first as a desquamative pneumonia, or appear first as a peribronchitis or a tubercular laryngitis.

first stage of their development,* and may do it with such absolute certainty that it was this very observation which proved to me the existence of a primary tubercular inflammation. The more recent the process the more discrete are the tubercles, and the membrane has the appearance of being studded with infection tubercles. In a later period the whole thickness of the layer of exudation, even as deep as the serous tissue itself, appears as a granular thick cheesy mass. This is brought about partly by an increase in the number of lymphoma, and partly, particularly after a preceding change of the embryonal cells into vessel-walls and endothelium, by a participation in the proliferation, of the latter cells between the lymphoma and the lymphatics which bind them together. The lymphoma contain still more commonly the giant cells, and thus without losing the analogy they depart more and more from the structure of the normal lymph follicles and become pathological lymphoma.

Notwithstanding the above-mentioned clinical characteristics of this peculiar constitution so favorable to the development of tubercular pneumonia, it is altogether too little understood; and it depends on a clearer distinguishing of the peculiar activities of organization to work out a better understanding.

Since my first experience, which I had with a pericarditis, I have taken great pains to gain information as to the *frequency of the formation of lymphoma in embryonal* (granulations) *connective-tissue in general;* and have made fibrous exudations on the different serous membranes occurring under the most dissimilar conditions, common carbuncles, etc., the object of investigation. I can now give it as my conclusion that the richer in cells is the neoplasm as compared with the intercellular substance, so much the *more certainly are lymphoma to be found.* There being but few cells, however, is no proof of the absence of lymphoma; and I might even go so far as to declare that *scarcely a circumscribed embryonal connective-tissue*

* Bericht der k. b. Akad. d. Wissensch. Sitzung vom 13. 6, 1863, p. 69.

neoplasm ever occurs without some production of lymphoma. If we agree with what has been said, and are convinced that in tubercular inflammation the lymphoma immediately joins the normal lymph-follicles, we shall be led to the opinion—an opinion based, however, on long-continued observations—that perhaps only the infection tubercles are distinguished by the presence of giant cells. There could thus be found not only an important histological difference between primary tubercular inflammation and secondary miliary tuberculosis, but also a foundation for the opinion that the giant cells owe their existence to the intrusion of cheesy substances, and hence bacteria, into the cell-protoplasm.

The fewer the lymphoma so much the more ephemeral do they appear to be; they are absorbed and disappear in the newly-formed connective tissue.

The cells which appear in new connective-tissue growths are in part spindle and stellate connective-tissue and capillary vessel cells, and in part spherical bodies whose indifference on the one hand to spindle and stellate cells is very evident, and which on the other hand show themselves as the first germs of lymphatic endothelium; these spherical bodies (cytoid) can both be and remain lymph corpuscles, that is, pus cells, which are produced within (*endogen*) the endothelial or connective-tissue corpuscles. It is not without interest to watch the coming up and disappearance of this connective-tissue new growth, composed sometimes only of the first-mentioned cells, those connected with fixed tissues, that is almost without pus cells, sometimes, however, almost entirely made up of the latter kind with slight development of the tissue alongside: it is also particularly interesting to see that only those organizations are connected with lymphoma which are very rich in tissue-forming cells, while in those which run chiefly to purulent infiltration the lymphoma are mostly wanting. I said before, that the richer in cells the new formation of connective tissue, as compared with the intercellular substance, so much the more

certain are lymphoma to be found; but we only get the full force of the remark if we remember that when there is a purulent infiltration we can scarcely speak of a formation of new connective tissue and of intercellular substance. Notwithstanding the difference, each runs into the other and exchange places, from which circumstance the purulent infiltration always gives a much more unfavorable prognosis, as in the last-mentioned forms of peribronchitis.

According to these facts we can define the *constitution in question as one in which there is a tendency in the organizing activities of an individual to respond to a slight irritation, by an inflammatory exudation extraordinarily rich in cells.* We have seen that with the abundance of tissue-forming cells the formation of lymphoma necessarily goes hand in hand, and the occurrence of a tubercular inflammation is accordingly explained.

The *particular irritation* in the blood and tissue juices which leads to the development of tubercles is then, in this case, *constitutional*, while in miliary tuberculosis it is the product of infection. Admitting this, we can no longer demand the existence of an infection for the production of lymphoma in tubercular inflammation. The lymphoma is only the expression of a quantitive change in the organizing activity of an organ, caused by some external irritation, while in acute miliary tuberculosis they are the expression of an auto-infection, and only the local process, connected however with the growth of the lymphoma, is inflammatory. *Tubercular pneumonia* is then and remains only a *higher grade of genuine desquamative pneumonia*, and this in turn is only intelligible as the localized expression of a general disease. This general disease is the peculiar constitution which, if we wish to form a scheme according to the degree of intensity, sometimes stops in its inflammatory exudation with the formation of spindle and stellate cells (called in the lungs pure genuine desquamative pneumonia); sometimes, however, it goes farther and adds to this a proliferation of the lymphatic endothelium (cheesy pneumonia);

and this again carried to a higher degree is accompanied by a production of lymphoma (tubercular inflammation), and may, under very unfavorable conditions, finally run into a purulent infiltraton (purulent peribronchitis with lobular suppuration).

In order more fully to characterize the constitution we must cite the *tendency of its exudations to caseation;* a tendency which is inherent in the cellular new growth. Besides the local constriction of the capillaries, this tendency may be caused by these vessels being feeble and narrow in their structure, and by a weaker force of the circulation from a weak contraction of the heart and from feeble respiratory movements ; which facts explain perfectly the absolute anæmia induced in the capillaries by the constriction of the exuberant growth of cells; also the necrosis and resulting cheesy degeneration.

Waldenberg (loc. cit., p. 167) declares "that the local abnormal condition of the thorax alone, fully explains the disposition to cheesy inflammation." I can, after all that has preceded, only accept the dictum with certain modifications. I am willing to acknowledge that the contracted chest, the feeble respiratory muscles, and the want of blood have an influence on the cheesy degeneration, but not on the particular form of inflammation which preceded it. You will certainly have met with many cases of the most pronounced anæmia (as after long suppuration of bone), with wasting of the muscles, and flattening of the chest, both as a consequence of the small amount of blood in the contained organs, and the limited movements in respiration, perhaps even accompanied by a bronchitis, without the development of either peribronchitis or tubercular pneumonia. Again you will remember cases where strong persons with well-developed chests have been suddenly attacked with cheesy pneumonia.

If we no longer have any reason for confounding miliary tuberculosis and tubercular pneumonia either as regards their pathology or their ætiology, then there ought to be

no confusion in respect to other anatomical points of resemblance; for instance, the fact that the yellow and enlarged tubercles of the lungs show small, punctiform, cheesy centres, which, if they are close together, assume a marked resemblance to cheesy pneumonia, and from the lack of cicatricial formations even become very commonly centres of infection. We ought from a scientific stand-point to rely entirely upon the cause for a differentiation; for the cheesy masses resulting from a tubercular pneumonia may lead to local and general tubercular infection; though this is a rare condition, since the condensed masses developing around the caseous product simultaneously with the degeneration, hinder the infection. Another reason is that the purulent products of inflammation and all portions of tissue in any way necrosed from anæmia, which have not the remotest connection with tubercular inflammation or with the hypothetical constitution, undergo caseation and lead to infection.

In order to remove, in opposition to tuberculosis as a result of specific infection, all specific character from tubercular pneumonia, which is at best only a higher grade of desquamative pneumonia, many authors would perhaps like to exchange the name "tubercular inflammation" for "*scrofular inflammation;*" and in so doing might acquiesce in Virchow's wish to call the tubercle, caused by the injection thus produced, a *heteroplastic scrofulide.* In opposition to this I must reply that by so doing, not only is nothing gained, but much is lost. We should have to oppose the fact that a tubercular infection may result from caseous products, but not from a tubercular inflammation; and further, by changing the name tubercular inflammation into scrofular we would to a certain extent give up and forget the histology of the process. We must accustom ourselves to connect *nothing specific with the name tubercular inflammation.*

These ideas I made known * in substance, only differently

* Ber. üb. 280 Sekt. p. 58, 68.

expressed, as early as 1856; and it was evidently on them that Niemeyer based his deductions concerning consumption. I had then, however, not made the distinction between the cheesy products of a tubercular inflammation and one which was not tubercular. The elements on which I then laid great stress, and which could all be included in "a general diminishing of the blood supply and enfeebling of the force of the circulation," which, as I remarked before, probably play their part in the cheesy degeneration of the products of tubercular inflammation, are the only conditions which can produce necrosis and cheesy degeneration, and that generally with the aid of either a mechanical or chemical cause. I say the only conditions, that is if we *except the hypothetical constitution*, that is, excepting desquamative pneumonia, cirrhosis, cheesy or tubercular inflammation.

Cheesy centres appear in the lungs after severe attacks of typhoid, exanthematous diseases, and pyæmia, after very large exudations, hæmorrhages, chronic suppuration, spermatorrhœa, prolonged lactation, etc., processes which are all accompanied by a general diminishing of the blood supply and enfeebling of the circulation. As local predisposing causes may be mentioned, emboli and thrombi in the capillaries, and compressions of the lung by pleuritic exudations. The caseation of the lung occurring under these circumstances, and the formation of cavities consequent upon a constriction of the tissues by the cheesy friable masses pulling on one another, are much more commonly found, as is the case also with foreign bodies, in the lower than in the upper part of the lungs. Atheroma of the bronchial arteries, which generally begins at the origin in the thoracic aorta, is also worth mentioning in this connection; also the stenosis of the cardiac mouth of the pulmonary artery.*

* Among the cases which Lebert has collected, the one published by me in 1856 (Ber. üb. etc., p. 60) was overlooked. The stenosis of the pulmonary branch in cirrhosis (Beermer), as well as the constriction of the roots of the lungs by indurated exudation, also belong to this class.

These two, the cheesy masses from tubercular inflammation and those caused by anæmia and debility, should not be confounded with each other.

Besides the results of difficult microscopic examination, there are other grounds for differentiating. The tubercular pneumonia is a diffuse lobar process, while the caseous masses resulting from anæmia and debility are always circumscribed, single or multiple, and if connected with foreign bodies, which have been forced into the bronchioles, are lobular. Tubercular pneumonia always begins in the apexes of the lung, while the other process may begin on many different points in the lung, but most commonly in the lower lobes. Tubercular pneumonia is always more or less progressive, and consequently its curability is extremely doubtful; the caseous masses from the other process are, however, when once placed, quite fixed in their original position, and their curability increases with the improvement in the anæmia and general strength of the body. Tubercular pneumonia around the cheesy spots always shows more or less of a commencing or fully formed cirrhotic thickening; the cheesy centres from anæmia and debility, however, since they do not have their origin in parenchymatous inflammation, always directly adjoin, as a lobular necrosis, the healthy lung parenchyma. We always see this form of lobular necrosis resembling most nearly the purulent liquefying and cheesy degenerating lobular suppuration of peribronchitis purulenta. It is, however, to be distinguished from this by microscopic examination, and by the condition of the neighboring tissue, or else they are indistinguishably mixed together.

As to their ability to cause infection they are all on the same level. The masses resulting from anæmia and debility which have no cirrhotic envelope, and on the same ground those resulting from the lobular necrosis of purulent peribronchitis, give, as I have said before, much greater danger of infection than the tubercular inflammation.

LECTURE XII.

Pulmonary Consumption.

We have now reached the final lecture of our course. I have expanded my subject as widely as seemed necessary to accomplish the purpose I had in view; although the amount of material is so immense that it might be almost indefinitely extended.

In conclusion, allow me to give you a short recapitulation, and to correct some of the prevalent ideas concerning pulmonary consumption. In fulfilment of a promise made in my last lecture, I must also add a few remarks on ætiology.

By the expression pulmonary phthisis, an expression having a terrible significance both to the profession and to the laity, we understand an advancing destruction of the repiratory organs, and, still more important, a decline and wasting of the whole body dependent upon the destructive lung lesion. Sometimes the emphasis lies more on one element, again on the other; and we may say that the more rapid the course of the phthisis the more does the stress lie on the destruction of the lungs, and the slower it proceeds the more prominent is the decline and wasting of the body.

In my lectures I have already brought to your notice quite a number of diseases which have the credit of leading to consumption. As the most important process of all, the foundation, the causa proxima of phthisis, stands forth *parenchymatous* or *desquamative pneumonia*, with its differences in course, grade, and form. Immediately following this are the closely related forms of *peribronchitis*. The

other inflammations of the lungs which I have described, as compared with these, are not only very far behind, but their causative relation is even denied.

I would like to rectify a few propositions which have been introduced into the pathology of phthisis. For example, Laennec declares that there is but one form of phthisis, the tubercular. If we include acute miliary tuberculosis under the head of phthisis, then it can be distinguished as phthisis from infection, and be received entirely in accordance with Laennec's idea. Of the other forms, which can be grouped under the head of "*inflammatory phthisis*," tubercular pneumonia only would be included by Laennec's definition, and all the other affections of the respiratory organs, such as chronic fatty degeneration, cirrhosis, cavities from hypertrophic bronchietasis and purulent peribronchitis would be altogether left out, although they too bear equally important relations to phthisis.

Infectious phthisis and inflammatory phthisis, as regards frequency of occurrence, bear the proportions of 8 to 100. It would be a great error to conclude with Laennec that phthisis never originates in an acute or chronic pneumonia, for desquamative pneumonia as well as peribronchitis with its lobular pneumonia, is the basis of the very frequent inflammatory phthisis. Both forms are included under the head of "pulmonary consumption;" for the connecting link which they both have in common is desquamative pneumonia, which would also include acute miliary tuberculosis.

They are distinguished by the fact that *infectious phthisis never produces any destruction of tissue, while the inflammatory scarcely ever runs its course without ulceration.* From this it might be said that inflammatory phthisis is the only true "consumption of the lungs." The connection is, however, clear, insomuch as the infectious phthisis can be added to the inflammatory (phthisis combinata, Waldenburg), the necessary predecessor of infection—cheesy remains of a previous inflammation—even existing in the lung.

Laennec's proposition must then suffer a modification: *phthisis is either primary and inflammatory or secondary and due to infection. The primary inflammatory form only can be a constitutional disease;* it is sometimes more acute, sometimes chronic.

If now we examine Lebert's proposition that "chronic disseminated pneumonia covers the whole field of consumption," we find the same to be true of it. It is not comprehensive enough to include the multiplicity of inflammatory forms with which you are already acquainted, and also the numerous combinations which they make with each other. By the terms "chronic and disseminated," the very important "acute and diffuse" inflammatory forms would be more or less excluded from being considered as the foundations of phthisis.

Though the study of the anatomical relations of phthisis may be very difficult, its ætiology is still more involved. In this connection we meet with another proposition of Laennec's, viz. that consumption is a *constitutional* disease. You may here recall what, when speaking of tubercular pneumonia, I said about constitution, and may very properly apply what was then said to the present case. To be sure our conception of the phthisical constitution is not fully formed; but we do know that it is characterized by a tendency not only to tubercular pneumonia, but particularly to desquamative (parenchymatous) inflammation of the lungs; and even more than that, from the tendency to the production of pus, we know it has a tendency to purulent peribronchitis.

We may find in the fact that purulent peribronchitis, as well as the non-purulent pure desquamative pneumonia and cheesy and tubercular desquamative pneumonia, may each occur alone and independently, a motive for separating inflammatory phthisis and its accompanying constitution into two divisions: one of these to be distinguished by a *formation of tissue* (under this head we must rank both the destructive and the non-destructive forms of desquamative

pneumonia), and the other to be marked by a *formation of pus*—under this head is to be placed purulent peribronchitis alone. Both these forms, which are rather subacute in their course, carry with them the idea of constancy and incurability. From them in turn are developed the chronic forms, such as fatty degeneration, cirrhosis with or without the production of cavities, lobular necrosis and caseation, and nodular peribronchitis. We must also add a third form of phthisis, a somewhat transitory form which is produced after severe diseases, losses of the fluids of the body, or great losses of strength, by the resulting anæmia and enfeebling of the circulation.

The constitution gives us, however, only one factor in the development of phthisis, that is, the latent phthisis. In order that it may really be aroused into activity, there is necessary an impulse from an external cause, in other words *an exciting cause.* I have thus far in my lectures only sought to give you the anatomy and pathology of this constitution, and have quite passed over its cause. I will therefore give you a few words on this point. Whether an external cause is capable by the extreme intensity of its action of producing phthisis in a person not marked out by this constitution as being peculiarly disposed to it, that is in a perfectly healthy organization, will be pointed out as we go on. It must act in such a case by causing a parenchymatous irritation and inflammation of the respiratory organs, as I have already shown.

In general, taking cold, and particularly climatic influences, are assigned as the causes of phthisis; and the succession of events is generally supposed to be first taking cold, then a bronchitis followed by catarrhal or croupous pneumonia, which leads to the phthisis. Before I enter upon a discussion of the casual relations of climate and atmosphere, I must examine more closely the recently much-discussed question, as to whether a catarrhal or croupous pneumonia, or an acute or chronic bronchial catarrh, are capable of producing phthisis.

Niemeyer has blamed Laennec with having introduced a very dangerous maxim into pathology. I must now, unfortunately, in turn, lay the same blame on Niemeyer; for I know of scarcely a greater pathological error than his conclusion that catarrhal and croupous pneumonia as well as chronic bronchial catarrh leads to phthisis. It all depends on the false supposition, that because the products of these inflammations (puriform masses of mucus and croup exudation) can undergo cheesy degeneration in the alveoli and bronchioli, they therefore represent the condition of cheesy pneumonia, and lead through this to necrosis and formation of cavities.

It shows an entire ignorance as to what cheesy pneumonia really is. Catarrh and croup are superficial processes, by which the capillary circulation is never interfered with; that is, anæmic necrosis and formation of ulcers cannot possibly be its results. The lung framework can, it is true, be in the highest degree œdematous, but is never (except in rare complications of croup, in purulent infiltrations and formation of abcesses, and in gangrene) seriously involved; consequently these products can only undergo fatty degeneration, scarcely ever caseation, and may be entirely absorbed. If from some mechanical cause they should here and there by chance be left in the lung, we need not fear either ulceration or infection, for the alveolar parenchyma and the walls of the bronchi come out uninjured from the previous process. When speaking of catarrhal and croupous pneumonia, I called your attention to the fact that those acute inflammations have nothing to do with cheesy pneumonia, and therefore nothing to do with consumption.

Chronic bronchial catarrh, also considered by Niemeyer as leading to phthisis, if it does not produce a mechanical emphysema does not affect the lung proper at all. I entirely agree with Laennec when he says that consumption never arises from a neglected or protracted catarrh. Niemeyer's idea rests principally on his having considered as one the superficial catarrh and the desquamative pneumonia or

peribronchitis. Both of these diseases may, it is true, be accompanied by catarrh, though this is not necessarily the case, as the so-called perforated tubercles (the lumina of a bronchus free from all secretion and the walls thickened) in peribronchitis nodosa or caseosa clearly enough prove.

The circumstance of a so-called bronchial catarrh being very long protracted should have led them to refer the appearances to a more deeply seated process.

If now the influence of climate does not provoke phthisis through a catarrhal or croupous bronchitis or pneumonia, the question presents itself whether it may not create this terrible disease directly. Statistics and geographical diffusion come partially to the aid of my previous explanations, by proving that *catarrhal affections of the lungs are commoner the farther we go from the tropics toward the higher degrees of latitude, while on the contrary and in opposition to the generally received opinion, the most northern portions of the earth show a certain immunity from consumption, while in the tropics consumption shows its greatest frequency and runs its course most rapidly.** The same holds true as to elevation; for while high plateaux and mountains are the home of catarrhs and bronchitis, a height of 2000′ above the sea-level may be considered as the limit of consumption. But we must also admit a certain dependence for phthisis as well as catarrhal affections upon atmospheric influences. A complete knowledge of the mode of action of the active elements of the atmosphere is still *in futuro*. We possess, as yet, no satisfactory explanation of "taking cold" founded upon exact investigation, and can only say that among these active elements temperature and humidity must be admitted as most important factors. On the other hand, the giving off of heat and water from the body, through the lungs, skin, kidneys, and mucous membranes, is of great importance; for the continuous process of compensation, if it cannot follow

* See Hirsch. Hist. geogr. pathol. II. p. 2, 55, 74.

the changes in the external atmosphere, must give occasion to great disturbance.

Unfortunately this reasoning partakes more of the nature of a scientific hypothesis than of a certain pathological fact.

As regards temperature, it is quite certain that it is not the mean temperature of a place which gives a measure of the frequency of catarrh and phthisis, but the sudden and frequent changes do the harm, by overcoming the compensatory activities of the body. It would be wrong to limit the action of the changes in temperature to the respiratory organs. Irregularities in the loss of heat and water are much more likely to produce a general disturbance, which can then be localized in any separate organ, preferably in those which happen to be in the highest state of functional activity at the time when the irregularities occur. For example, in very violent exertions the respiratory organs would be in a state of high functional activity, during digestion it would be the intestinal canal, and at another time perhaps the kidneys.

If now a person who is exposed to the influence of cold has no peculiar tendency to disease, he may get off with a catarrhal bronchitis or a pneumonia or croup; but if he has the peculiar phthisical constitution, then the cause which produced the cold will make a deeper impression and produce a parenchymatous inflammation. This may be either desquamative pneumonia or peribronchitis, in a word, phthisis. Taking cold, by itself alone, can never cause phthisis; it can only awaken the phthisical tendency to primary or renewed parenchymatous inflammation. We can understand how Cormak could declare that "the inhalation of cold air has never caused a single case of consumption since the world began." *The temperature of the air and its sudden changes must therefore be admitted as exciting* (*procatartic*) *causes of inflammatory phthisis.*

The relations which the dampness of the atmosphere bear are somewhat different. We must not leave out of

sight that an atmosphere very poor in water may give to our sensations an impression of being very damp; while an atmosphere thoroughly impregnated with vapor may seem to us as being very dry.* Most of the situations in which consumption is very frequent are distinguished by a very moist atmosphere, while those enjoying almost an immunity from its attacks are marked by extreme dryness. This idea † has very lately received very important confirmation from a report by Buchanan.‡ He found the moisture of the soil, on which depends, if not entirely, at least to a very great degree, the dampness of the air, to be a very important cause of consumption among the population which lived on it. He showed that *by drainage nearly all danger of consumption could be removed from a given place.* Elevated plains and places with a porous soil can be easily and effectually drained, and may thus come to offer an immunity from consumption. Clayey soil, on the contrary, and low-lying situations, are only with great difficulty, and even then improperly drained, and are consequently the homes of consumption. We may admit, then, that a constant and high degree of moisture in the air and soil do create the phthisical constitution; and only the absence of exciting (procatartic) causes, sudden changes of temperature, can compensate for such unfavorable surroundings. The moisture and warmth which surround us have thus great pathological importance in respect to the disease of which I am speaking, for they are at the foundation of both the factors of phthisis, the peculiar constitution and the exciting cause.

I must now call your attention to external causes which act on the respiratory organs by direct mechanical and chemical means; that is, they act similarly to foreign bodies falling into the air-passages. I alluded to them in my second lecture, but only so far as the intensity of their action was sufficient to create a superficial catarrhal irritation. The question, however, is whether all of them, or if not all,

* Hirsh. loc. cit., p. 10. † Same, p. 77.

‡ J. Simon in 10th Report of Med Office of Privy Council, 1867.

which ones, are able to provoke a parenchymatous irritation. I told you before that the products of catarrhal and croupous inflammation which remain in the finer bronchi, in very rare instances, distend the bronchioles; and then, a peribronchitis ensuing, a thickening results, and thus becoming encapsulated, they may within the capsule undergo cheesy and chalky degeneration. This process is therefore simply a circumscribed parenchymatous inflammation, which neither depends on the phthisical constitution nor on a continuous external cause.

Still more important, though, from their being acute, more transitory in their effect, are decomposing substances which, being drawn into the lung on inhalation, act locally in a chemico-mechanical manner. They act not only superficially on the mucous membranes, but even go so far as to set up destructive processes in the lung parenchyma itself, producing ulceration and gangrene. As a result we may then have absorption, and the blood being infected, the whole body may be poisoned. Under this head may be mentioned masses of croupous membrane detached from the larynx and trachea, portions of diphtheritic substances the contents of pulmonary cavities, fecal masses from stercoraceous vomiting, fungi from fœtid bronchitis, pneumonomycosis, etc.

Of late the importance of *blood extravasated into the air passages* has caused a great deal of discussion. Niemeyer has proclaimed the same as to the great danger of hæmoptysis, which he falsely proclaimed for bronchitis. Together with this most alarming assertion, he has made another which is much more comforting, and which is naturally enough much more acceptable both to the profession and to the laity. While, on the one hand, he tells us that, contrary to Laennec, who held it to be always the sign of an already existing consumption, bleeding from the lungs is the cause of a future phthisis, on the other hand, he tells us that hæmorrhage from the bronchial mucous membrane, which at the bedside can with great difficulty be

distinguished from true pulmonary hæmorrhage, is to a certain degree harmless. I have known many practitioners who were very much inclined to regard every hæmoptysis as being bronchial in its origin, and thus lulled themselves and their patient into a false security as regards the future. If they saw themselves mistaken, then the case was used to prove the origin of phthisis in hæmorrhage. For myself, I am forced to deny *in toto* that blood which has been extravasated into, or has run into the air cells, can undergo cheesy degeneration, and form a cheesy pneumonia. Aside from the fact that the cheesy masses of desquamative pneumonia are clearly formed from alveolar epithelium, and certainly never from blood corpuscles, though a few may get mixed up with the cells, there are strong theoretical grounds against it. I cannot see why blood in the alveoli should be more dangerous than the products of a catarrhal or croupous pneumonia: it can be absorbed quite as well and even better than they, for the previous morbid process in the enclosing walls, as the advocates of that opinion insist, are at that time still wanting. On the same grounds, the exuded blood can scarcely take on cheesy degeneration; for the presupposed conditions, the absolute anæmia of the alveolar walls, and consequent necrosis, are entirely wanting. The hæmorrhage is therefore, as in croup, either the result of an acute genuine or necrosing desquamative pneumonia, and is then capillary in its origin, and the sputa have blood mixed with them, or it is the result of the separation of a slough, the formation of an acute cavity, or the softening of a cheesy mass and the opening of a large vessel in consequence. If it comes under the last division the sputa are found to be streaked with blood, or else a larger or smaller quantity of pure blood is thrown out. No one will be willing to believe that a hæmorrhage can come by chance; and no one can prove that exuded blood can produce an inflammatory irritation and lead to desquamative or to cheesy pneumonia and formations of cavities. The blood aspirated into the

lung parenchyma is certainly admitted by the alveolar epithelium which desquamates and degenerates, as I explained when speaking of catarrhal pneumonia. These facts are established by the investigations of Waldenburg, Sommerbrodt,* Perl, and Lippmann.†

Clinical observers were induced, at least so it appears to me, to consider hæmorrhage as the cause of a future cheesy pneumonia, by its commonly occurring in an apparently healthy person, but in whom there had existed a small necrosis, which could not possibly be diagnosticated, and which, in general, did little harm. They also overlooked the fact that a desquamative or cheesy pneumonia may come on as suddenly as a croupous pneumonia, and that it may spread quickly over either a whole lung, a lobe, or a part of a lobe.

The sudden physical changes in the auscultation and percussion is often referred entirely to the infiltration of blood, while it really belongs to the inflammatory infiltration; or where it really can be referred to the blood, there it is not mentioned that it is soon entirely absorbed without leaving any cheesy pneumonia behind. I do not deny, however, that bronchial and pulmonary hæmorrhage do occur without parenchymatous pneumonia. Cases are not unfrequently met with where persons do spit up blood who were neither ill at the time of the hæmorrhage nor become so later. These cases depend as a rule either on diseases producing a dissolution of the blood, or on a congestion in the capillary circulation dependent upon some affection of the respiratory organs themselves, or on a cardiac lesion which produces capillary ectasis in the lungs. Niemeyer concedes the common genetic connection between spitting blood and inflammatory pulmonary diseases; and he does not deny the occurrence of hæmorrhage in latent pulmonary diseases (consumption), or what is the same thing, in a consumption occurring later, but without being able to make clear the genetic connection.

* Centralblatt, 1871, No. 43. † Virch. Arch. 51. Bd. p. 552.

Notwithstanding the opinions of such weighty authorities as Niemeyer, Virchow, Hoffmann, Rindfleisch, etc., I believe that neither catarrhal nor croupous pneumonia, nor chronic bronchial catarrh, and still less pulmonary or bronchial hæmorrhage can ever, without the peculiar constitution, and without a real occurrence of parenchymatous pneumonia, lead to cheesy pneumonia. When seeking for the cause of phthisis, the question always turns, not upon the cause of the caseation of the bronchial and alveolar contents, but upon the necrosis and cheesy degeneration of the alveolar and bronchial walls, and of the lung-framework. This question is either not answered at all by these authors or it is entirely overlooked. The fact has been observed that it does occur, but no inquiry has been made to discover the reason; and so they have been content with the opinion—an opinion, however, in the highest degree irrational—that the pressure of their contents on the alveoli and bronchial tubes is the cause of their necrosis.

Quite different from the action of the products of the mucous membrane produced in the air passages, and from the action of effused blood, is that of *foreign substances* which in the shape of *dust are mixed with the inhaled air.*

These substances, when inhaled, even where the constitutional tendency does not exist, by themselves alone and simply by their continuous mechanical irritation excite inflammation. This inflammation is not limited in its effects to the mucous membrane and its epithelium, but, by penetrating deeper, produces a destruction in the lung parenchyma, and is thus connected with processes which end in cicatrization and thickening or in necrosis and ulceration.*

It is undoubtedly true that a healthy, strong individual may throw off a severe attack of this disease, but only on the condition that the cause is entirely removed. In those cases where the cause continues in operation the disease spreads more and more, and finally runs into a true phthisis.

* See Lecture II.

If this be the course of events in a naturally healthy person, you can easily imagine how much sooner an organization imbued with the phthisical tendency will answer to the foreign penetration by an inflammation rich in cells, and, even though the exciting cause be removed, the individual will perish from consumption.

Such admixtures of dust with the air are found in workshops, manufactories, mines, etc. The operatives in our manufactories furnish the largest percentage of phthisical cases. Hirt * has quite recently published a very careful collection of cases of disease from inhalation of dust occurring among workingmen, and shows a frightful number (80 per cent.) as suffering from phthisis.

The most dangerous occupations are those of the file-cutters, goldsmiths, bronze-workers, stone-cutters, manufacturers of French millstones, steel and brass grinders, cutters of diamonds, precious stones, glass, and porcelain; polishers with sand-paper, hare-wool cutters, braiders in hat factories, flax-combers, horse-hair pickers, shoddy pickers, etc. I refer you to Hirt's book as well worthy of study.

The experiments lately published by Sommerbrodt † are of great importance in this connection.

He concludes that foreign bodies cause inflammatory phthisis, not altogether by direct contact with the lung parenchyma, that is, the dust-like substances which are mixed with the air and inhaled do not produce parenchymatous inflammation only when drawn into the alveoles, but an action on the larynx and trachea may be quite sufficient to cause phthisis. He explains it in this way: the mechanical irritation of the larynx and trachea causing an inflammation, this inflammation extends as a true peribronchitis until it finally reaches the lung parenchyma. He also says that the alveolar parenchyma is not involved in severe diseases of the larynx with infiltration and ulcera-

* Diseases of Workingmen, vol. i.

† On the dependence of phthisical pulmonary disease on primary affections of the larynx. "Arch. für exper. Pathol. etc."

tion, as has been supposed, by the inhalation of the detritus of the ulcer, but explains this in the same way, by an extension of the inflammation. In his experiments he passed rings of wire through the larynx and trachea, to produce the necessary irritation.

I have given dampness of the atmosphere and soil a high rank among the external causes tending to create the phthisical constitution. The so-called "foul air" contains, besides dust, another very important element in the production of phthisis. I refer to the mixture of great quantities of *unhealthy gases*. I need not mention them individually, but they are all important in this connection, that is, in their relations to the development of the phthisical constitution. It is an incontrovertible fact that consumption has a direct connection with the density of the population. Towns, and particular quarters of a town, are therefore particularly dangerous. Still worse are places which are shut in, and where proper ventilation is impossible, as narrow, contracted habitations, workshops, manufactories (in this case both the dust and the foul air work together), barracks, prisons, and schools; and these are all the worse the less number of cubic feet there are per individual living or working in them. Of the three in a thousand ($\frac{3}{1000}$) of the population who die of consumption, the fearful number of 12–15 per cent. are those who are placed under conditions such as I have just mentioned.

You can easily understand why more poor than rich persons die from phthisis, and why the disease is more frequent in our climate in winter than in summer. If we add to these conditions, as affecting the poor, the insufficient and often spoiled food, the want of clothing, the great strain on both body and mind, the often-occurring sorrow and care, insufficient exercise (an element which can, as well as anxiety, sorrow, etc., be applied equally to the rich), we are not surprised that the poor furnish the principal number of the cases of phthisis.

We have already recognized bad air, defective nourish-

ment, and imperfect clothing as factors in the ætiology of scrofula, and from the similarity in the causes may infer a similarity in result. Besides, scrofula, syphilis, and, perhaps, other not well-understood constitutional diseases (*e. g.*—Addison's disease), are to be considered as belonging in the same rank.

Finally, we find one of the most marked characteristics of the phthisical constitution to be its property of hereditary transmission. This may be recognized in at least one-third of the cases. So much for the ætiology of phthisis.

Before I close, you must allow me, even at the risk of making my lecture too long, to make a few remarks on the diagnosis, and on the implication of the whole organization in the disease.

The *diagnosis of the acute form* is difficult sometimes on account of the local, and again from the general symptoms. The general symptoms are sometimes so very prominent as to make it necessary to use extreme caution in differentiating it from other acute general affections.

The resemblance between the symptoms of typhoid fever, complicated by a catarrhal or croupous pneumonia, and the symptoms of acute miliary tuberculosis, is often so very marked that a differential diagnosis is impossible. I have seen five cases of scarlatina, "sine exanthemata," complicated with cyanosis, and such severe chest lesions, that the physical signs pointed to an acute miliary tuberculosis, or at least to the accompanying desquamative pneumonia, until the scaling off of the epidermis forced us to a change of opinion. It must not be confused with parenchymatous myocarditis, for not only may the implication of the brain through acute œdema give a great resemblance to typhus fever, but the progressive development of brown induration of the lungs and the dependent cyanosis render a distinction from miliary tuberculosis hardly possible. Again, necrotic desquamative pneumonia may begin with the symptoms of typhoid, and the physical signs in the beginning being those of a croupous pneumonia, only the continuance of the disease

long past the usual crisis (twenty days) may first show us our error. We may make other mistakes if we declare a purulent peribronchitis to be only a simpler catarrhal bronchitis.

The *diagnosis of the chronic cases* is rendered more difficult if the portions of lung tissue only partially filled with air (fatty) and those which are empty (cheesy and cirrhotic) are very small in circumference, for then they escape the most careful physical examination. This is still more the case with a nodular peribronchitis which appears under the garb of a discrete or grouped tuberculosis. Only large cheesy or cirrhotic nodes, widely-spread fatty degeneration, and cavities (generally bronchial cavities which have increased very much) can be quite easily distinguished; and then no doubt remains as to the trouble with which we have to deal.

If we can learn nothing concerning the family history of scrofula in childhood, repeated attacks of pulmonary inflammation, coughing up of blood, etc., and know only of the tedious course of the trouble and the frequent relapses, and if we are not willing to await the secondary emaciation and marasmus of the body as proofs, then there remains really nothing for us to do in order to find out easily the true nature of an attack accompanied by frequent cough or hacking but to make repeated and frequent examinations of the apices of the lungs. In the apices, the first and most important physical signs are met with. The so-called apex catarrh is certainly not without reason a very much-to-be-dreaded symptom. This catarrh, taken in connection with diminished height of the point of the lung (Ziemesen), the murmur of the subclavian heard above the clavicle during expiration (Rühle), are commonly the first recognizable objective signs. Lancinating pains, flying or continuous, blood in the sputa, even true hæmoptysis belong among the very earliest symptoms. A duller percussion sound, slight sinking, and diminished expansion confined to a spot, very soon follow. To properly value these signs, and

to be able to decide in the very first stage as well of the acute as the chronic forms, we certainly very often wish for other supplementary signs.

Among the aids which we can call to our assistance are the appearance of tubercles in the choroid coat of the eye in acute miliary tuberculosis, recognizable by the ophthalmoscope. I attach still greater importance to a *microscopical examination of the sputa.* I refer less to the long well-understood sputa of the later stages (sputa globosa, lanuginosa), which contain molecular masses, schizomyceten and fungi, as well as elastic fibres, which point out with certainty the existence of a necrotic pneumonia with a formation of cavities, but much more the nature of this sputa. I described this in my lecture (V.) on genuine desquamative pneumonia, which is the foundation, not only for acute miliary tuberculosis, but for tubercular pneumonia—I might say for the phthisical lung in general. Up to the present time these examinations have been nearly overlooked, and there are now many physicians who do not even imagine that such an examination can be of any use in a diagnostic point of view. This depends largely on the fact that the anatomical knowledge of the processes in the lungs was very limited, and that which after the tubercle is the most important, the lung parenchyma, was entirely overlooked.

If we omit the admixture of blood which may occur in pneumonia or myocarditis, in the acute exanthemata, in typhus and other severe diseases, just as well as in phthisis, if we omit also the puriform mucus, a product of the secondary catarrhal bronchitis, which may occur with desquamative pneumonia, then we have in the sputa, first, lung epithelium, more seldom ciliated epithelium, in a state of fatty degeneration (granular cells), here and there sprinkled with black pigment; second, lung epithelium from the thickened walls glistening, and with proliferating nuclei; third, lining epithelium, in a state of myaline degeneration: all of which show the existence of desquamative pneumonia. By microscopic examination of the sputa

we can diagnose not only acute miliary tuberculosis, but also genuine pure and cheesy pneumonia, even in the very first days of the attack. We can in this way recognize the very smallest spots, which escape entirely auscultation and percussion. We can also show that the apex catarrh is but the expression of a desquamative pneumonia, instead of an ordinary bronchiolitis, and this even at a time when no cavities have been formed.

For purulent peribronchitis, unfortunately, there are no certain microscopic signs. There are only signs of destructive processes, molecular masses of purulent sputa, generally without any lung epithelium, and the very easily discoverable elastic fibres from the bronchi. These latter, with the physical signs of bronchial catarrh, give us some guidance; but the appearance of fever, very commonly alternated with chills, give a much more certain ground for diagnosis. The quantity of myeline in the sputa is a distinguishing mark of the chronic forms, a rule to which nodular peribronchitis makes the single exception, that is, when it occurs alone; most commonly it escapes diagnosis entirely. The other ordinary signs of phthisis you are already quite familiar with.

With our ideas concerning the affection of the constitution according to which it has its seat in the lymphatic system we cannot be surprised if it finds expression, not simply in the lungs, but also in other organs.

One of the commonest expressions of the disease is tubercular inflammation in the intestinal canal (82 per cent. of phthisical patients suffer from diarrhœa); and the stretching of Glisson's capsule, and in part the pale yellow-color of the skin, depend perhaps on the same cause. Sometimes the colon alone is attacked; again it may be the ileum, or the whole intestinal tract, even the stomach, and it may extend thence as far as the mouth. The larynx and trachea are very commonly attacked, for 15.5 per cent. suffer from hoarseness or loss of voice.

One of the commonest effects is a deterioration of the blood; anæmia distinguishes chronic consumption. By the

decrease in the respiratory surface, that is, in the pulmonary capillaries, fewer blood-disks are oxidized, and the amount of arterial blood is thus decreased. From these simple circumstances it comes to pass that not only the arterial blood, but the whole mass of the blood is lessened in amount, and the body is correspondingly weakened. This may occur even where the supply of food and its digestion in the stomach and intestines is quite complete or even superfluous. This follows from the fact that the arterial blood is not only quite necessary to the maintenance of the body and for the accomplishment of all the metamorphoses in the body, but also from its serving the purpose of an excitant to the activities of the blood-forming organs.

Subjective and even objective abstractions of bodily heat, particularly from the surface and the extremities, increased irritability, even an incredible sensibility and sensitiveness of the nervous system, characterize and accompany the advancing affection.

The causes of the anæmia and wasting are thus seen to be quite different from those operating in other diseases. While here, aside from the disease of the lymphatic system, the injury to the respiratory organs is the cause, in other diseases, in stenosis of the stomach for example, it is the limited supply of food; in cancer of the liver it is the decrease in the quantity of liver tissue necessary to the formation of the blood, etc. When catarrh of the stomach and intestinal canal, with or without ulceration or other degenerations, is superadded, depending on the constitutional affection, then only does pulmonary phthisis come to resemble other causes of inanition, since then nourishment can be used without any benefit to the organization. Hæmorrhage from the lung helps on very rapidly the diminution in the blood supply.

Besides anæmia, another very important element has great influence on the general nutrition. If a person having the normal amount of blood in his body is suddenly attacked by a desquamative pneumonia, then, before a noticeable diminution in the general amount of the blood can

take place there is necessarily an imperfect decarbonization of the blood, and we see as a result cyanosis.

From anæmia and cyanosis together arises the *general fatty degeneration.* This is found in 54 per cent. of phthisical cases, taking the acute and chronic together. It shows itself distinctly in the liver, kidneys (with albuminuria), in the heart (by thrombi here and there), and in the external muscular system. In the last-mentioned system it has a very peculiar interest, when it appears as an *acute parenchymatous myositis*, with painful swelling (so-called rheumatism) over one or another joint, and even wanders over the whole body. It begins generally with severe fever. It lays claim to a very particular interest, from its not always occurring in cases where phthisis already exists, but very often in cases where, up to the time, the person has been perfectly healthy, as the first symptom of a pulmonary phthisis, in the beginning scarcely credible, because not yet recognizable. It runs through the well-known histological changes (fatty degeneration, hyaline or waxy degeneration), even up to muscular atrophy; and there may occur on isolated spots an induration of the skin and even muscular hypertrophy as a result.

The often solid sputa, the *profuse sweating* not less than the diarrhœa, which, in consumption, tends not a little to wasting and exhaustion, have similar foundations; that is, degeneration of the mucous glands of the throat and respiratory passages, and of the glands of the intestine and skin. I have found the fatty degeneration in the intestine attacking not only the glands, but also the villi, and creating in places such a friability of the tissues that they seemed as if ulcerated off. Until now, this ulceration from softening has not been recognized, and I imagine that an increasing growth of fungi has a part in the play.

Special observations have long ago pointed out the *clubbing of the terminal phalanges of the fingers*, and it has been ascribed to the hinderance of the circulation, particularly in the peripherical parts. It seems to me very probable

that this lesion is connected with scleroderma, which certainly very commonly begins in the finger ends, more seldom in the toes, and spreads from these points further over the body.

This disease ends almost always in phthisis. Both of these diseases, scleroderma and clubbed fingers, are analogous to pulmonary cirrhosis, and depend on hypertrophy of the connective tissue.

I have been able in a few cases of commencing scleroderma to predict pulmonary phthisis, and this has begun only after two or three years.

In respect to the amyloid degeneration I have already spoken at length in a previous lecture, and will only add that in phthisis it can occur not only in the lungs, but also in the liver, spleen, kidneys, intestines, and larynx, in fact, in most of the organs, and that it is followed by hydræmia.

With pulmonary cirrhosis and a slight degree of anæmia we have an *hypertrophy* of the *right side of the heart*, and exceptionally a fatty degeneration. In a higher degree of anæmia there is an atrophy of the heart. The first of these changes in the heart accounts for palpitation, asthma, cyanosis, nutmeg liver, congested kidney, hyperæmia of the mucous membrane of the stomach and intestine, chronic pachymeningitis with formation of osteophytes, atrophy of the brain with dropsy ex vacuo, and general dropsy, as is not very seldom met with.

You know that consumptives breathe very frequently and superficially, and that dyspnœa is excited on the slightest exertion. You also know the various causes for this phenomenon: anæmia, cyanosis, fatty degeneration of the cardiac muscles, fever, increased irritability, and this in many cases without pleuritis or extended destruction.

I need not, however, go through all the symptoms of phthisis. It is not necessary to mention the *fever*, which is often so severe as to resemble at the time a true typhoid. The continuance of the hectic fever does not depend so much on relapses and extensions, that is, on the consump-

tion, as upon the very certain absorption of decomposing masses by the blood. The intermittent chills belong either to tubercular infection or to purulent peribronchitis, the latter offering the principal opportunities for putrid infection.

If now you review the symptoms of pulmonary phthisis,* you will see that it is not possible for me to agree with the clinical pictures which Niemeyer has drawn, for they depend on false conceptions.

In the *first* place, he declares that a croupous pneumonia in which the fever lasts over the second week has run into a cheesy pneumonia. I have sought to show that this never occurs; but that such a case was a desquamative pneumonia from the very beginning, and should have been diagnosticated in the very first days by a microscopic examination of the sputa.

Secondly. He holds that pulmonary hæmorrhage is primary, and the cheesy pneumonia the result. I have endeavored to point out that a pulmonary hæmorrhage cannot cause a cheesy pneumonia; and that in these cases, where phthisis is observed to follow hæmorrhage, the beginning of the former must be referred back to a point before the advent of the first bleeding.

Thirdly. He presupposes an acute bronchial catarrh. I

* I might propose to seek the clinical appearances, previous to the following schema, in which naturally only the phthisical processes are included, and catarrhal and croupous pneumonia omitted:

I. ACUTE.

1. *Phthisis from infection* (acute miliary tuberculosis).
2. *Inflammatory phthisis.*
   a. Lobar (pure desquamative and necrotic pneumonia) acute pulmonary cavities.
   b. Lobular (purulent peribronchitis).

II. CHRONIC.

Inflammatory phthisis.

   a. *Lobar* caseation, fatty degeneration, and cirrhosis: bronchial cavities.
   b. *Lobular* caseation and nodular peribronchitis.

III. PHTHISIS COMBINATA.

have effectually shown that phthisis cannot arise from catarrh, and that in this case one of two deep-seated and severe diseases, either desquamative pneumonia or peribronchitis, have been confounded with the catarrh.

Fourthly. He pictures to us a chronic catarrhal pneumonia. But such a condition does not exist. What Niemeyer called by this name was generally pulmonary cirrhosis or cheesy lobular pneumonia (from peribronchitis).

Finally, allow me a few words, since it has been particularly requested, concerning *therapeutics.* As this subject does not properly belong to me, the remarks will be very brief.

What we have to do by our therapeutics is, on one hand, to improve the disposition, the tubercular constitution, and on the other, to improve the already advancing disease, or to extinguish it, and protect the patient from the influence of new exciting causes.

Rühle is right when he says that all the trouble of the pathological anatomist has accomplished nothing, for it is no more consoling to die nowadays of chronic pneumonia or amyloid degeneration with dropsy, than it was in times past to perish by tuberculosis or consumption. The pathological anatomists, however, cannot now be reproached, for they have endeavored to do their share of the work. The burden has been shifted upon the clinicians and therapeutists, who, notwithstanding the exact knowledge which is furnished them of the nature of the disease, are able to save only a very few of these patients from inevitable death. I, as pathological anatomist, do not, however, disparage the therapeutists, and greatly admire, for instance, the wonderful advance made by Buchanan, his masterpiece. He not only attempts to fight against one of the causes of phthisis, the constitution, but he has actually conquered. A few more such results, and therapeutists may congratulate themselves.

To bring about an improvement in an acute attack already begun (I mean by this, not the inaccessible infection, but the inflammatory form), that is, an attack of

acute cheesy pneumonia, or purulent peribronchitis, it is not only necessary to limit the extension of the process, but to cause it to take on a lower grade of action and to become chronic. This succeeds less often in younger patients, who are particularly liable to be snatched away by acute inflammatory (*floride*) and infectious phthisis. If the extension of the affection is limited, we may hope from that circumstance that the youthful age may be sooner accessible to constitutional changes than adult age. Most of the victims of phthisis are between the ages of thirty and fifty, though the disease may occur in the six-months-old baby and in the octogenarian. Further, it becomes chronic more easily in females than in males (3 : 2), among the rich than among the poor, and among those who live in good climates, etc.

If, happily, chronicity is finally attained, the extension limited, and the disease reduced to the lowest grade, the genuine pure desquamative pneumonia, then we still have to fear not only the formation of cavities and a slowly creeping extension of the disease, but also with reason a tubercular infection (Niemeyer). If infection does not take place, then everything depends on the possibility of improving the constitution and removing the direct and exciting causes.

These, in brief, are the principles of the treatment of this disease. The acute forms must be treated symptomatically, as is more or less the case in all fevers.

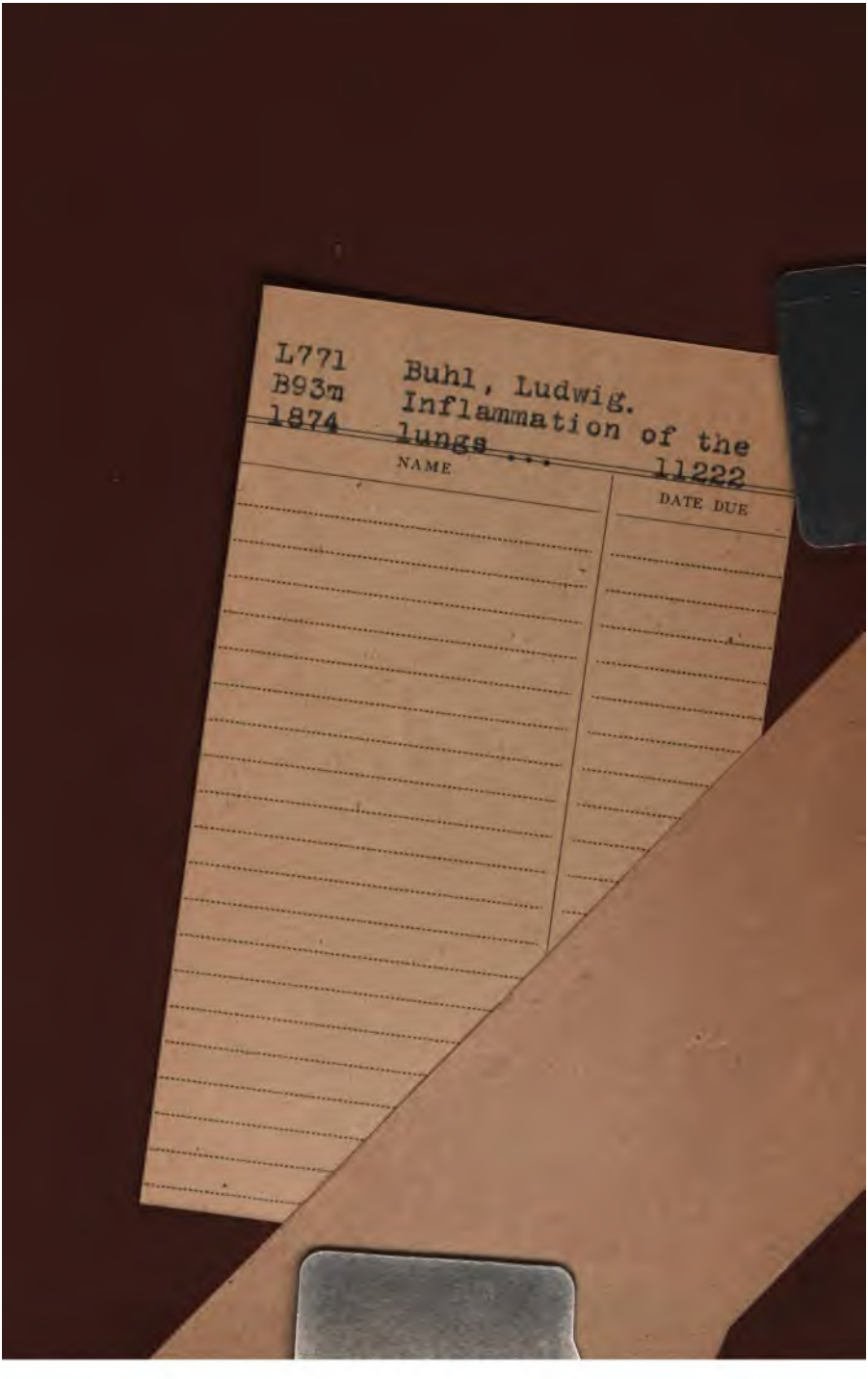
For commencing or already well-established chronicity, the (I might almost say) *principal cure is good air.* There must be plenty of air, and it must be free, properly changed, without dust, rather dry, and not subject to great changes of temperature: this latter is particularly necessary in a medium degree of moisture. The residence should be in a spot sheltered from the wind and on well-drained soil, with large, airy rooms. South of the Alps (in Europe), if possible, and during the summer, an elevation of from 2,500 to 3,000 feet should be sought. In the spring and autumn this may be changed for an elevation of from 1,500 to 2,000 feet.

In winter the patient may dwell either near the sea, or at an elevation of from 500 to 1,000 feet.

Nearly as important as good air is proper food. It should be easily digestible; and not meat only, but milk and eggs should be used; and in some cases the patient should be restricted to them and the hydrocarbons, which are not to be undervalued, such as the amylacea, sugar, butter and other fats, and I generally choose the finest kind of olive instead of cod-liver oil; and if severe cough and fever do not counterindicate it, generally recommend as a drink either beer very rich in malt, or slightly warmed red wine. We must not leave out of sight the benefit of proper clothing and a mind as free from care and pressing duties as possible. All dangerous employments should be given up. The patient should exercise properly, but should never carry it to the point of fatigue. In calm and warm weather he should sit out of doors as much as possible. All these points carefully considered in the selection of a residence, and all the suggestions carried out to the smallest detail, will in the end often reward us with most gratifying results.

There still remains a wide field for the employment of a rational therapeusis. The indications for the particular medicines must be gathered from the symptoms. I would particularly call your attention to the use of external irritation of the skin. This may be by dry or moist friction, and should last a very short time, from a half to one minute, or we may employ spirits instead. I would also recommend in some cases the application of a greater amount of heat, as by cloths dipped in oil, or compresses wet in tepid water and covered with gutta-percha, or the latter substance alone. The medicines most useful are quinine, iodide of potash, Fowler's solution, seldom iron, and still more seldom morphia.

THE END.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